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CHRONIC ATROPHIC DERMATITIS OF THE VULVA[1]

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UNDER this broad descriptive terminology we wish to describe a condition the nomenclature of which has aroused considerable discussion. It was first described under the term of "kraurosis vulvæ" by Breisky in 1885, and each succeeding author has been so dissatisfied with this limited descriptive term as to introduce a new one. Thus we have an array of names for this condition, the more common of which are leucoplacia (Morris), leucoplacic vulvitis (Taussig), leucokraurosis (Graves), and pruritus vulvæ. Although the catchy term "kraurosis" will probably remain attached to this condition, we suggest the use of a broader, more descriptive name—chronic atrophic dermatitis of the vulva. This simplified nomenclature is in line with the general tendency to use simple descriptive terms. Furthermore, this term is broad enough to include early cases of dermatitis of the vulva which may develop into typical kraurosis.

Fig. 1. Photograph showing the early hypertrophic stage. Note the marked edema of the labia minora and majora and the presence of superficial blebs and petechial hemorrhages. There is slight involvement of the perineum.

Fig. 2. Photograph showing the second stage. Atrophic changes can be noted in the disappearing labia minora and narrowing of the introitus. The clitoris is entirely covered over by the adherent atrophic preputial folds. Note the characteristic whitening of the mucosa and the numerous superficial petechial hemorrhages. Note likewise the sharply demarcated involvement of the lesion.

Fig. 3. Photograph showing the third stage. All signs of elasticity have disappeared and the vaginal orifice is greatly constricted and rigid. The labia minora have disappeared completely, only the urethral orifice and the preputial folds being present. The perineum is involved in a typical triangular shape.

KRAUROSIS, LEUCOPLACIC VULVITIS, LEUCOKRAUROSIS

These three most common terms are all used specifically by a few authors, but interchangeably by most of them to designate progressive pathological atrophic changes in the vulva. Undoubtedly, different patients present themselves with various pictures of this condition. However, we agree with Taussig and Graves that these various clinical manifestations represent different stages of a progressive disease which, if allowed to go untreated, will ultimately develop leucoplacia and possibly carcinoma. It is, therefore, perhaps simpler to think of the entire condition under the broad name of chronic atrophic dermatitis of the vulva, reserving kraurosis and leucoplacia for various stages of the condition.

INCIDENCE

The disease is very infrequent and cases are rarely seen in general practice. In large gynecological clinics, however, a fair number are encountered. In 9,682 gynecological patients examined for the first time at the University of Chicago Clinics, chronic atrophic dermatitis of the vulva was encountered in 23 patients—an incidence of 0.24 per cent. Probably many cases seen in their incipiency, when pruritus is the only symptom, remain unrecog-

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nized until the pathological condition becomes well marked. The number of cases recorded in the literature is not a true index of its frequency.

ETIOLOGY

Chronic atrophic dermatitis of the vulva is distinctly a disease of the menopausal or postmenopausal years. It rarely involves young women during their active sex years. Parity apparently plays no rôle as the incidence is probably as frequent in women who have had children as in nulliparous women. Cessation or waning of the ovarian function undoubtedly is an important factor in this condition. The average age in Taussig's large series of cases was 49 years, and that in our group was 52 years. The condition occasionally occurs in young women, particularly those suffering from abnormalities of ovarian function. These cases, however, are rare and when they do occur they merit careful study as to the etiology. Of the patients in this group, 7 still had regular or irregular menses, and 16 had ceased menstruating.

Inflammation is probably the most important, constant etiological agent in the production of this condition. A fertile field for inflammation is provided in the physiological atrophic changes which occur during or after the menopause, as is pointed out. Szász, Veit, Taussig, Graves, and Smith, all emphasize the inflammatory process associated with this condition. Pruritus is a constant symptom in this condition, and several of the authors look upon it as an etiological agent in the production of the disease. It is hardly fair to assume that it is the only factor because there must always be some cause for the pruritus. The constant itching leads to trauma in the form of many small wounds and bruises which heal slowly and with difficulty because of the infection and the continued irritation. In the slow healing process a piling-up of the superficial layer of epithelium occurs. This may account for the keratosis and whitening of the superficial epithelium, giving the characteristic picture of leucoplacic vulvitis.

PHYSIOLOGICAL ATROPHY OF THE VULVA

Normally, following the onset of the menopause, a slowly progressive atrophy of the sex organs takes place. This is most marked in the uterus and the ovaries, and less marked in the vagina and the external genitalia. During the early years of the menopause, this atrophy of the external genitals may be barely visible, becoming more marked with advancing age. The atrophy consists in a shriveling-up of the labia minora and majora, and the vagina. This shriveling-up process is aided by the loss of fat in these parts. The skin may become smooth, thin and glistening, and the vaginal introitus may become greatly narrowed. The vagina, likewise, undergoes even more extensive atrophic changes, which we have studied recently. The trauma of intercourse, examination, or any simple manipulation may cause these atrophic areas to become bruised or cracked, thereby producing petechial hemorrhages which may lead to soreness or itching. So far, these changes may all be considered entirely physiological, as the result of a loss of certain ovarian function. This condition has been called "simple kraurosis" because of the characteristic shriveling-up process, but it is far better to ascribe it to the end-results of the cessation of certain physiological ovarian activity.

A sudden cessation of ovarian activity, resulting from an artificial menopause induced by radiation or surgical castration, may hasten the development of the physiological atrophy of the vulva.

As a rule, this physiological process requires no treatment as it produces no symptoms. However, when it occurs in a middle aged woman, particularly one in whom an artificial menopause has been induced, the narrowing of the introitus may make coitus difficult, exceedingly painful, or even impossible. It may be necessary to provide additional room at the introitus by some surgical expedient, such as the operation devised for a vaginismus.

When this physiological atrophy is marked and repeated trauma causes fissuring, bruising, and petechial hemorrhages, it is possible for these bruised and fissured areas to become infected. The atrophic skin is no longer a good barrier to the introduction of even the usual variety of organisms which are always present, and the infection may become more or less chronic. Trauma, petechial hemor-

rhages, and infection cause irritation of the superficial nerve endings. This irritation is usually interpreted by the patient as itching which is mild at first and becomes more marked as the condition progresses. Itching calls for scratching on the part of the patient, thereby causing more bruises and trauma to the delicate vulval structures. Thus, in a period of months or years, a true chronic atrophic dermatitis of the vulva develops, predisposed by a normal physiological process.

Three or four stages of the disease are usually described; however, the process is progressive and no clear-cut picture of each stage can be seen. They rather tend to merge into one another until the well developed atrophy appears. Various stages coincide and may be seen at the same time.

First stage. In the earliest stage, the inflammatory process may be most marked. The entire vulva is swollen, red, bruised, and painful. The labia minora and majora and preputial folds stand out prominently due to the swelling. In some cases the edema may be so marked as actually to cause small superficial blebs of the labia minora and majora. The skin appears stretched, glistening, and at times almost parchment-like in its transparency. Superficial wounds, abrasions, and petechiæ may also be present. The condition involves the labia majora and minora and the introitus, ending at the mucocutaneous junction and the perineum (Fig. 1). If the infection is marked the inflammatory process may extend out laterally over the skin of the thigh, causing fissures in the groin. Burning and itching are marked and the skin is actually painful to the touch or the pressure of clothing. This early stage may last for weeks, months, or years, depending on the acuteness of the process and the amount of discomfort to the patient. If the inflammation is marked, the usual soothing treatment of the skin will cause the process to subside somewhat.

Second stage. The atrophy begins to manifest itself grossly in the second stage. The skin itself appears thickened, indurated, and leathery in character due to increasing thickness of the keratin layer. It has lost its elasticity and the usual suppleness, so that an ordinary pelvic examination in a multiparous woman causes pain. The color is a whitish to a mottled gray, with scattered pinkish or reddish areas caused by the petechial hemorrhages. These superficial hemorrhages in their absorption and organization go through the various stages of the usual subcutaneous hemorrhage, providing the various colorations to the vulva. The labial folds flatten out so that they may appear only as ridges. All trace of the labia minora may disappear. The preputial folds likewise flatten out or become completely obliterated (Fig. 2).

The characteristic distribution of this condition is most interesting. The changes are usually limited to the non-hairy portion of the vulva and are sharply demarcated at the mucocutaneous junction with the vagina. The perineal skin is usually likewise involved, extending down to the anal region. Occasionally, the atrophic process extends about the anus posteriorly to it. This specific limited area of the diseased process lends credence to the endocrine origin of the condition.

Third stage. After a period of years, most cases are finally characterized by this last stage, during which many of the patients present themselves for treatment. The skin over the involved area becomes smooth, glistening, semitranslucent, and parchment-like. Minor trauma causes it to crack, fissure, and bleed. It is pearly-white or bluish-white in color, with petechial hemorrhages in the various stages of absorption. All signs of elasticity have disappeared. The vaginal orifice becomes greatly constricted and rigid. The labia minora have completely disappeared. The preputial folds have become ironed out and only the urethral orifice stands out anteriorly. The extent of the perineal involvement varies in different patients. Here the skin assumes an appearance typical of the vulvar changes. This stage of the disease, likewise, is progressive, although it may appear unchanged for several years at a time. There does not seem to be a complete spontaneous retrogression of this condition, although there may be long periods during which the symptoms as well as the course may remain quiescent (Fig. 3).

Leucoplacic areas often develop in chronic atrophic dermatitis as single or multiple discrete plaques involving the inner surface of

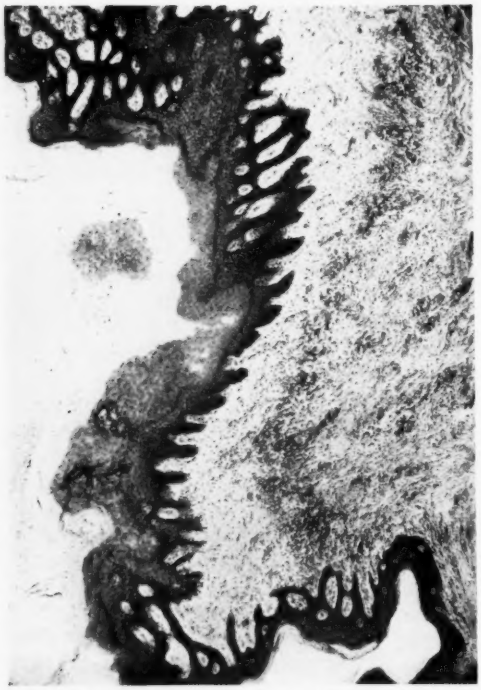

Fig. 4. Photomicrograph of the hypertrophic stage showing the increased thickness of the superficial layer of keratohyalin and marked elongation of the papillæ. There are no abnormal changes in the basal layer of cells, but beneath it there is marked edema and a characteristic layer of inflammatory cells. ×45.

the labia majora and minora, the area over the perineum or around the clitoris. The latter is most frequently involved. These leucoplacic areas may cause the most intense itching. They are grayish or bluish in color, with a definite thickening of the skin or mucous membrane. Fissures or ulcerations may occur about these regions.

There are rare cases in which the changes described are confined to one side of the vulva or remain localized to such an extent that one may speak of a localized leucoplacia. However, in the large majority of patients, this condition symmetrically involves the entire vulva and extends into the perineum.

HISTOPATHOLOGY

In the histological picture of this condition one may easily follow the progress of the disease. Few specimens have been taken in the incipient state because surgery has been rarely resorted to until the condition is far advanced. It is possible, however, to recognize two distinct stages—the hypertrophic and the atrophic stages. Taussig, however, has pointed out correctly that in one specimen all stages of the disease may be found. Thus, the progressive atrophy does not continue at an equal pace over the entire area.

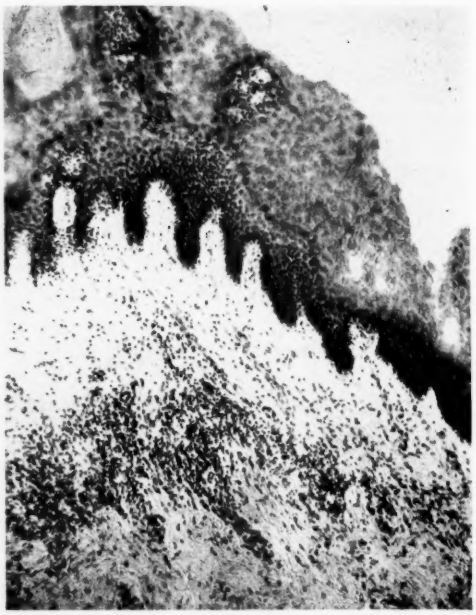

Fig. 4A. Photomicrograph showing the characteristic changes in the hypertrophic stage. ×90.

In the early *hypertrophic stage*, after the acute inflammation and edema have partially disappeared, one finds marked elongation of the papillæ and an increased thickening of the superficial layer of keratohyalin (Figs. 4 and 4A). The basal layer of cells shows no abnormal changes. All of the cells stain deeply and show marked granulation of the cytoplasm. This granulation gives the same appearance to these cells as is normally found in the intra-epithelial zone of cornification, or Dierck's layer seen in the vaginal epithelium (Fig. 5). Normally, this layer becomes most marked in the vaginal mucosa just about the

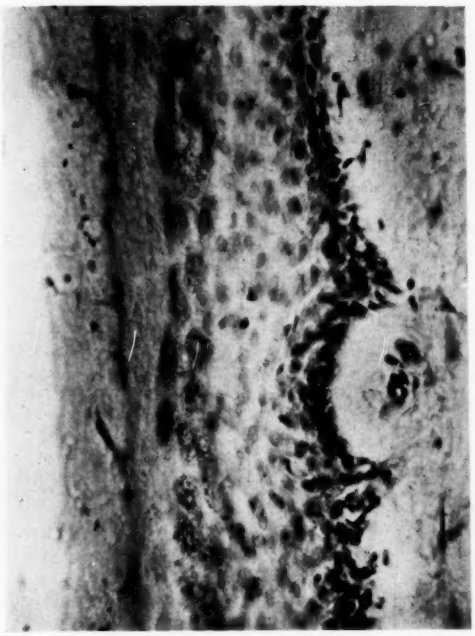

Fig. 5. Photomicrograph of the hypertrophic stage showing a layer of large cells filled with granules between the basalis and functionalis. This layer resembles the intra-epithelial zone of cornification or Dierck's layer seen in the vagina. ×300.

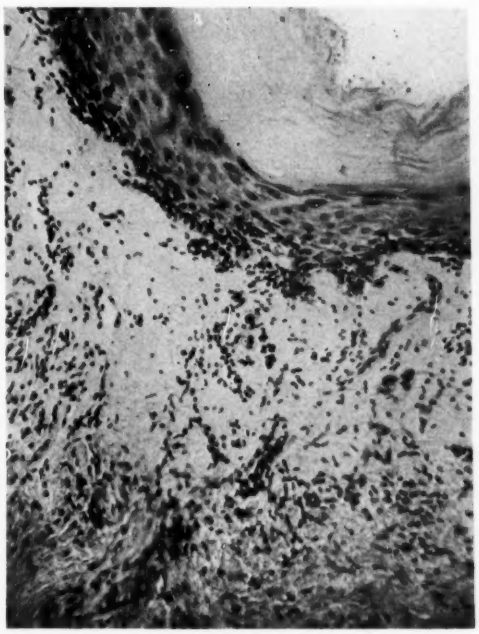

Fig. 6. Photomicrograph of atrophic stage showing the relationship between basalis and greatly thickened functionalis. Note layer of large cells containing granules of various sizes beneath these two layers. The basal layer of cells appears frayed and irregular. ×170.

time of ovulation. Apparently it is associated with the building up of the functionalis. The keratin layer increases considerably in thickness. The most marked changes are found just beneath the squamous epithelium. There is a marked cellular infiltration of the connective tissue consisting of inflammatory cells. Round cells predominate, although polymorphonuclear leucocytes, plasma cells, and other wandering cells may be seen. These inflammatory cells occur in smaller and larger foci and at times densely infiltrate the subsquamous zone. Increased vascularity and edema are likewise noted. Marked changes occur in the elastic fibers early in the disease. They may be entirely absent between the papillæ.

In the late *atrophic stage*, the squamous epithelium has become greatly decreased in thickness. In some areas it consists of only four or five layers of cells. The papillary projections have almost disappeared. The basement layer of cells appears ragged, irregular in outline, and stains poorly. In the individual cells, liquefaction necrosis may be seen. The superficial keratin layer has steadily increased so that it now appears as thick or thicker than the basalis (Fig. 6).

The most interesting changes may be seen in the dermis beneath the squamous epithelium, where there are large edematous areas in which few, if any, living cells may be found (Fig. 7). These may be due to a transudate of serum and a liquefaction of the connective tissue of this layer. As a result of the edema the elastic tissue has become separated from the epidermis and now appears frayed and irregular beneath this edematous area. In places the elastic tissue fibers seem to be swept up into irregular bundles like driftwood. It is likely that they have lost their continuity, ruptured, and retracted to form

Fig. 7. Photomicrograph showing marked atrophy of the basalis. Beneath this layer there is extensive edema in which there remain small islands of cells composed of leucocytes, plasma and mast cells. There is a marked absence of elastic tissue in this edematous zone. This elastic tissue appears beneath it. ×95.

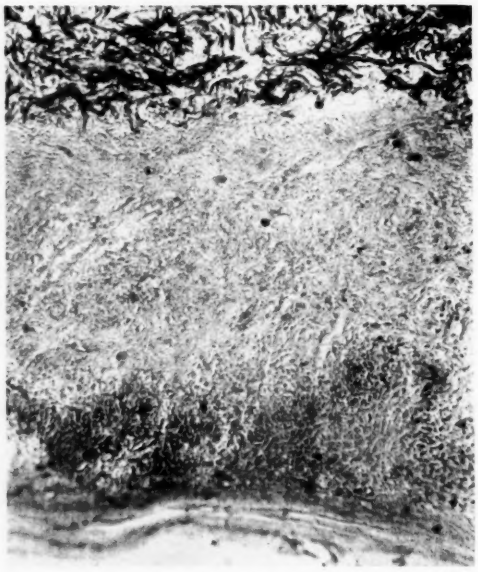

Fig. 8. Photomicrograph of a section stained with Wiegert-van Giesen's elastic tissue stain. The edematous layer beneath the basalis shows almost a complete absence of cells. Beneath this layer there appear fragmented strands of elastic tissue stained black and swept together like driftwood in this irregular zone. ×155.

these unusual accumulations (Fig. 8). The zone of inflammatory cells is now present beneath the edematous layer, consisting chiefly of mononuclear and polymorphonuclear leucocytes and mast cells. All of the wandering cells may be seen. In the dermis is a marked absence of sebaceous glands of all types. Fewer blood vessels than appear in the hypertrophic stage may be seen. There is relatively little change in the deep layers of the cutis and the subcutaneous tissue, except for some atrophy.

Histologically, leucoplacic areas represent hypertrophic processes and may be indistinguishable from kraurosis vulvæ. There is a marked hyperkeratosis, the elastic tissue is frayed and broken up, and a diffuse infiltration of inflammatory cells occurs, consisting chiefly of lymphocytes which are perivascular in distribution. The small vessels in the subepithelial layer show varying degrees of narrowing due to thickening and fibrosis of their walls. Again, we wish to call attention to the fact that the atrophic and hypertrophic stages may be found in the same specimen. Thus, this progressive process does not involve all parts with equal rapidity.

SYMPTOMS

Pruritus, with rare exceptions, is a predominant symptom, and was the chief complaint of all the patients in this group. The duration of pruritus may be noted in the tabulation of these cases. The itching may be slight at first, causing little inconvenience. It steadily becomes worse so as to upset the normal nervous equilibrium of the patient. The itching may be worse at night, leading to marked loss of sleep (Tables I and II).

In a few instances, burning rather than itching is the most common complaint. This is most marked during the acute stage of the disease, particularly when the element of infection predominates. If there is marked excoriation about the vulva, burning on urination may be troublesome. Likewise, if the

TABLE I

Present age Menopause	Symptoms	Areas involved	Treatment			Remarks
			X-ray (Total R-units)	Medical	Surgical	
61 45	Genital itching, burning—2 years	Labia minora, majora, perineum, anus	846 Aug., 1933	Calamine lotion	Vulvectomy May, 1934	No. 81908. Results of vulvectomy good. Other therapy gave no relief. Slight itching of anus still persists.
57 33	Vulval, anal itching, burning 10 years	Vulva and perineum	1,200 Jan., 1933	Lotions Salt packs	Vulvectomy June, 1934	No. 67688. Results of vulvectomy good. Other therapy gave no relief. Stage 3.
48 47	Vulval itching 2 years	Labia minora, majora, perineum, vulva	None	Calamine Phenol lotions	Vulvectomy Apr., 1934	No. 50538. Local therapy gave no relief. Partial vulvectomy. Itching has returned. Stages 2 and 3.
59 49	Vulval itching, burning after X-ray for carcinoma of cervix	Labia minora, majora, perineum, vulva	None	Calamine lotion	Vulvectomy Apr., 1934	No. 99003. Results of vulvectomy good. Stages 1 and 2.
56 46	Vulval itching, burning 3 months	Entire vulva, perineum	1378 Sept., 1930	Local therapy	Vulvectomy July, 1932	No. 26023. Felt fine 1½ years following radiation. Itching returned. Results of vulvectomy excellent. Stage 3.
62 47	Vulval itching, burning 2 months	Labia minora, majora, perineum, vulva	1145.6 Nov., 1930	Local therapy	Vulvectomy Mar., 1931	No. 6787. Radiation gave no relief. Results of vulvectomy excellent. Stage 3.
49 44	Vulval itching 10 months Pea-sized wart on labium 1 month (carcinoma of vulva)	Left labia, entire vulva, perineum	2502.3 June, 1933 After operation	Lotions	Vulvectomy May, 1933	No. 81523. No relief from local therapy. Results of vulvectomy good.
40 —	Vulval itching, discharge 9 years	Labia minora, majora, perineum, vulva	1015 May, 1933	Local therapy	Vulvectomy Sept., 1933	No. 59628. Symptoms aggravated following radiation. Results of vulvectomy excellent. Stage 3.
57 51	Genital itching, Severe irritation	Marked inflammation of parts extending to thighs, edema	Elsewhere	Amniotin	Vulvectomy Sept., 1934	No. 104449. Radiation given elsewhere. Amniotin therapy gave no relief. Patient hospitalized for treatment of infection and vulvectomy. Stage 1.

excoriation extends to the anus, painful defecation is the result. Many of these patients complain of dyspareunia and pain on examination. This is due to loss of elasticity of the skin, causing cracking and fissuring.

The progress of the disease is slow. Mild cases may persist for years without causing great discomfort. The intolerable itching eventually brings the patient to a physician. In some cases the progress is more rapid so that in the course of a few months marked atrophic changes develop. The usual antipruritic lotions and ointments give little, if any, relief.

DIAGNOSIS

The vulva should be examined under subdued or indirect light which will accentuate the outline and appearance of the involved tissue. A well developed case of chronic atrophic dermatitis of the vulva should not be difficult to diagnose. It occurs particularly in women at the menopause or postmenopausal period, thereby ruling out conditions causing pruritus in young women. On careful inspection the characteristic skin changes, previously described, will be noted and found to be well demarcated. In the early stages, however, considerable difficulty in making a diagnosis may be encountered. At times a positive diagnosis may not be made until evident atrophy occurs and typical kraurotic and leucoplacic changes are manifest. In the early stage, therefore, all conditions which may engender itching of the vulva and the perineum should be ruled out.

Infections of the vulva cause pruritus. The various pyogenic organisms may be associated with leucorrhea and inflammation. The vestibule, labia minora and majora, and perineum may be reddened and tender. Smear and culture examinations will identify the various pyogenic organisms. Therapy directed

TABLE II

Present age Menopause	Symptoms	Areas involved	Treatment		Remarks
			X-ray (Total R-units)	Medical	
71 51	Severe itching, redness, swelling of vulva 12 years	Labia minora, majora, perineum, vulva	Elsewhere	Local therapy	No. 103026. Prior treatment gave no relief. No clinic treatment.
47 46	Itching of perineum 5 years	Labia minora, majora, vulva	546 May, 1933	Castile soap	No. 82790. Radiation gave temporary relief. Stage 1.
61 56	Itching of vulva 8–10 years	Labia minora, majora, vulva	238 July, 1930	Local therapy	No. 2416. Radiation gave temporary relief. Suggested vulvectomy. Patient did not return.
50 49	Itching of vagina 17 years	Labia minora, majora, vulva, perineum	570 Aug., 1934	Local therapy	No. 45198. Condition improved. Patient did not return. Stage 3.
52 36	Itching of vulva 8 years	Labia minora, majora, vulva, perineum. Fissures extending into anal ring	1127 Mar., 1933	Boric powder	No. 76966. Marked relief for several months, but symptoms have returned recently. Stage 3.
40 —	Itching of labia 5–6 years	Perineum extensively involved	1284 Mar., 1932	Local therapy	No. 55791. Radiation gave relief for 1 month, but aggravated symptoms returned. Suggested vulvectomy. Patient did not return. Stage 3.
57 47	Severe itching of vagina, rectum 10 years	Labia minora, majora, perineum, vulva	None	Calamine lotion Phenol	No. 51832. Medical therapy gave temporary relief. Suggested vulvectomy. Patient did not return.
42 —	Itching of labia minora 4 years	Marked edema of vulva, perineum	915 Jan., 1933	Local therapy	No. 59738. X-ray therapy gave no relief. Advised vulvectomy. Patient did not return. Stages 1 and 2.
53 —	Itching of vulva	Labia minora, majora, vulva, perineum	None	Local therapy	No. 92648. Local therapy gave no relief. Advised vulvectomy. Patient did not return. Stage 3.
50 —	Itching, burning of vulva, discharge, pain 2 years	Labia minora, majora, vulva, perineum	None	Local therapy	No. 84544. No treatment given. Suggested hospitalization. Patient did not return. Stages 1 and 2.
60 53	Itching of vulva, pain, discharge, dysuria	Vulva, perineum	None	Amniotin	No. 109031. Improved under amniotin therapy. Stage 1.
50 32	Itching, discharge, dysuria—2 years	Labia minora, majora, preputial folds, perineum	600 Sept., 1934	Local therapy	No. 112544. Radiation made condition worse. Discontinued. Vulvectomy to be done.
40 —	Itching, discharge dysuria—1 year	Labia minora, majora, preputial folds, perineum	Elsewhere	Amniotin	No. 110465. Local treatment is being given. Amniotin therapy made condition worse. Hypertrophic stage.
19 —	Itching of perineum—12 years	Labia minora, vulva	None	Ichthyol ointment	No. 14130. Improved after medical treatment.

toward the eradication of the infection will result in a prompt improvement.

Yeast infection of the vulva is probably more common than is the consensus of opinion. It has been shown recently that it is almost always associated with diabetes. Superficial scrapings of the involved skin will show the organisms on microscopic or culture examination. Blood sugar determinations should be made in every case of vulvitis. Similar lesions elsewhere on the body may assist in making a diagnosis (Fig. 9).

Other fungous infections of the vulva may occur, usually associated with lesions in the groins. The characteristic skin lesion is usually present. Microscopic examination will reveal the fungus as the etiological agent.

Parasites, such as pediculi or pinworms, occasionally are responsible for pruritus vulvæ. In the absence of a specific skin lesion, other than the abrasions and trauma of scratching, careful examination of the vulval hair and the anal region should be made to determine whether or not parasites are present.

Neurodermatitis. Dermatologists recently have emphasized the importance of neurodermatitis as a cause for pruritus vulvæ and ani. The occurrence of the condition in young women with an absence of any characteristic local changes in the skin should suggest a neurogenic origin. The presence of a neurodermite elsewhere substantiates the diagnosis. This condition is usually found in the nervous, unstable individual.

Fig. 9. Yeast infection of the vulva, involving the labia, skin, and both groins. Superficial skin scrapings revealed the organism on microscopic and culture examinations.

Fig. 10. Photograph of a small polypoid squamous cell carcinoma of 6 to 8 weeks' duration superimposed on chronic atrophic dermatitis of the vulva.

Syphilis has been given as an etiological agent for kraurosis vulvæ by the older authors. It probably has no relation to this condition. However, a Wassermann test should be made on every patient.

Many other rare conditions may be responsible for pruritus of the vulva and should be considered in the differential diagnosis.

The development of an *epithelioma* of the vulva is always a possibility and may occur at any stage in the disease. Every patient should be followed carefully and a biopsy made of any suspicious lesion. The appearance of a nodule on the vulva in a case of chronic atrophic dermatitis is highly suggestive of a squamous cell carcinoma, inasmuch as over 50 per cent of these patients ultimately develop malignant changes. Chronic atrophic dermatitis of the vulva is the most important socalled "precancerous lesion."

PROGNOSIS

The disease does not tend to regress spontaneously, although long periods may elapse during which there is little noteworthy change. The great danger to the patient is the possible development of a squamous cell carcinoma. Kraurosis and leucoplacic vulvitis predispose to malignant changes in at least 50 per cent of the cases. Chronic atrophic dermatitis of the vulva may be termed a precancerous lesion, for in no other condition is a disease so often followed by malignancy. It may arise in the hypertrophic or atrophic stages (Fig. 10).

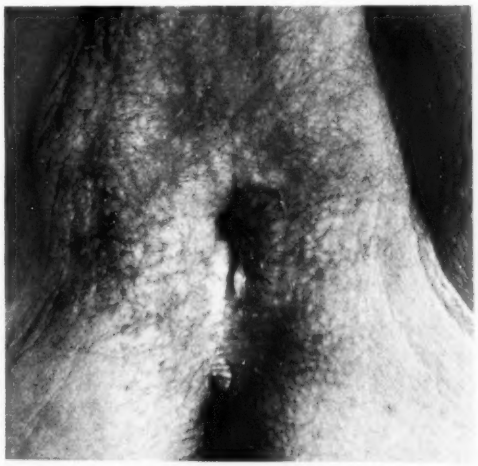

Fig. 11. Photograph showing the postoperative results of a vulvectomy performed 2 years previously. On examination the vagina easily admits the finger. The patient has had no return of her symptoms.

TREATMENT

Although many types of therapy have been advocated from time to time, surgery is the treatment of choice. In dealing with a lesion which is progressive in character and frequently followed by malignant changes, adequate surgical removal appears logical. A vulvectomy, varying in extent with the area of the skin involved, removes all source of danger of malignancy. The operation carries with it very little risk and may be performed on almost any individual. We have done a number of vulvectomies under local anesthesia, supplemented by narcotics, with entire comfort to the patient. The relief from the pruritus is immediate. Convalescence is usually uneventful. Healing will generally occur by primary union if there is no constriction of the tissues. In middle aged women a functional vagina may be restored, although in older women the normal vaginal atrophy precludes this (Fig. 11). Taussig reports a case of leucoplacic vulvitis in a young woman who gave birth to a normal full term child following a vulvectomy.

X-ray therapy has been given for this condition with some success. Early in the disease, before marked atrophic changes appear, radiation occasionally will give temporary relief from the pruritus. It does, however, recur and the progress of the disease continues unaltered. Most of our patients received radiation therapy with little or no relief, and were subsequently operated upon. Radium is not useful in the treatment of this condition.

The usual anti-pruritic lotions, salves, and ointments are of no benefit to allay the intolerable itching. Their efficacy is lost after several applications and the patient loses faith in their dispenser.

Hormonal therapy has been given with no apparent success. Future treatment along this line may eventually prove useful.

Learmouth has advocated sectioning the pudendal, perineal, and other nerves of the vulva to allay the itching in chronic atrophic dermatitis. The results of this method have not been uniformly successful. In removing the itching, the patient may be lulled into a sense of security while malignant changes are developing. The desirability of this form of therapy is open to question.

CONCLUSIONS

1. The present terminology—kraurosis, leucoplacic vulvitis, leucokraurosis, etc.—is confusing and unsatisfactory as it describes only certain phases of this condition. It leads to failure in making a diagnosis of the early stages of the disease prior to the development of the shrinkage of kraurosis or the white areas of leucoplacia. Chronic atrophic dermatitis of the vulva is a simple and descriptive term for the entire process in its various manifestations.

2. In a period of 5 years we have encountered 23 patients with typical chronic atrophic dermatitis of the vulva—an incidence of 0.24 per cent. Vulvectomy was done in 9 patients with uniformly good results. Various types of treatment were given, including radiation in the majority of the patients, with only temporary relief.

3. The disease is progressive and does not tend to regress spontaneously, although there may be periods of quiescence.

4. Surgical removal of the involved tissue is the only safe, logical, and effective treatment in alleviating the symptoms and arresting the progress of the disease.

5. The condition is to be regarded as a precancerous lesion, with an incidence of carcinoma in over 50 per cent of the cases.

6. Vulvectomy is further justified as a prophylactic measure against carcinoma of the vulva.

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PHLEBITIS, THROMBOSIS, AND THROMBOPHLEBITIS OF THE LOWER EXTREMITIES[1]

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PHLEBITIS, thrombosis, and thrombophlebitis are discussed together here because it is difficult to draw a sharp differentiation between the three conditions. Most phlebitides are associated with some thrombosis, and even the blandest thromboses provoke a reactive inflammation of the adjacent vein wall. The three terms, moreover, are widely interchanged, usage frequently reflecting the opinion held as to the relative importance of vein infection and thrombosis in the evolution of the picture. Thus, in the German literature, the term thrombosis is widely encountered, thrombophlebitis being reserved for the frankly infective forms of the disease. In France, on the other hand, where the teachings of Vaquez still prevail, phlebitis is the term most often employed. It is the purpose of this paper to discuss briefly the conditions characterized by vein inflammation and thrombosis under a single heading, in the hope that some of the vagueness and confusion surrounding them may be dissipated.

GENERAL CONSIDERATIONS

Thrombosis. Three chief classes of causes for thrombosis have long been recognized. These are changes in the vessel wall, slowing of the circulation, and alterations in the composition of the blood itself. The last named of the three probably represents the oldest theory. John Hunter enunciated the belief that inflammation of the vein wall was the necessary precursor of thrombus formation, and that the clot was always secondary. Virchow established the conception of thrombosis as an intravascular coagulation due to slowing and eddying of the circulation. If the inflammation played any part, it was merely that of slowing the blood stream, thereby permitting the clot to form. Zahn, some 20 years later, demonstrated that trauma to the intima of living vessels resulted in local thrombosis. He observed the deposition of white blood cells along the injured surface and differentiated between red and white thrombi, that is, between coagulation of the blood as occurs in extravasated or completely stagnant blood, and agglutination, which is the primary process in intravascular thrombosis. Hayem and Bizzozero recognized blood platelets as independent blood elements, and demonstrated that it was their rôle, rather than that of the white corpuscles, in the agglutinative process, which initiated thrombus formation. Further studies by Eberth and Schimmelbusch, Welch, Aschoff, and others have confirmed these observations, and have added materially to our knowledge of the mechanism of thrombus formation.

Although their relative significance is still widely disputed, it is now generally accepted that all three of the factors mentioned are involved, and that an interaction of two or more is probably necessary for the development of thrombosis in the majority of cases. In the thrombophlebitides due to penetrating injuries, or arising by extension in areas of suppuration, the inflammatory alterations of the vein wall are obviously the initial changes. Bland thromboses, on the other hand, arising after clean operations or deliveries, and involving vessels at a distance from the field of operation cannot so easily be explained on the same basis. Between these two extremes are the large number of phlebitides with thrombosis arising during the course of infectious states, following operation or in the puerperium, in which the possibilities of infection somewhere in the body are present, but in which there is no direct connection between its location and the site of the phlebitis. In these cases, it is difficult to state whether the vein becomes infected first, with secondary thrombosis, or whether secondary infection of a thrombus occurs, with subsequent involvement of the vein wall.

Slowing of the circulation is certainly one of the important causes in the development of thrombosis. This is attested by the relative

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infrequency of thrombosis in the aorta and larger arteries despite the most extensive sclerotic changes, even to calcification and ulceration of the vessel wall, whereas thrombosis of the veins is comparatively common. Postoperative thrombosis is practically never seen in children, and is rare in young adults. It becomes frequent during middle and advancing age, when changes in the heart and blood vessels permit slowing of the blood stream. The vessels most often affected in this type of thrombosis are the large veins of the lower extremities, those of the left side being much more frequently involved than those on the right, in spite of the greater frequency of surgical intervention in the right half of the abdomen. This selective localization has been accounted for on the basis of anatomical variations on the two sides, such as greater length and obliquity of the left common iliac vein, pressure of the right common iliac artery which crosses and compresses it against the vertebral column, constriction in the arterial loop formed by the left internal and external iliac arteries (Kistler), and pressure from the adjacent portion of the colon, all of which further favor local venous stasis. The frequent formation of thrombi in varicose veins, aneurisms, and similar locations where slowing and eddying of the circulation occur, further indicates the importance of this factor in the development of thrombosis.

Hunter showed, about a century ago, that complete stagnation of the circulation by means of aseptic and atraumatic isolation of a column of blood between two ligatures does not lead to thrombosis. This experiment has been confirmed many times, notably by Vaquez and by Baumgarten, and has been invoked as an argument against stasis being the sole cause of thrombosis. The invalidity of this argument lies in the failure to differentiate between thrombosis and coagulation. Coagulation, such as occurs postmortem or when blood has been shed, is a function of stagnant blood and depends upon the liberation of ferments from the cellular elements, which activate the thrombin in the plasma. This, in turn, converts the soluble fibrinogen into strands of insoluble fibrin. The formed elements are enmeshed in the fibrin network, the whole solidifying into a more or less homogeneous jelly-like mass. Thrombosis, on the other hand, is a phenomenon of blood which is flowing, albeit at a reduced rate, and consists of the agglutination of platelets along the vessel wall to form a homogeneous layer. Superimposed upon this mass is a settling out of white blood corpuscles. This gradual building-up of a white thrombus can occur only while the blood is still flowing. Fibrin formation plays no part in this picture, except as a secondary process when, as a result of the liberation of ferments from the agglutinated platelets and leucocytes, coagulation may occur at the periphery of the thrombus. As a matter of fact, thrombosis has been observed in blood rendered incoagulable by the addition of hirudin, in the experience of Zurhelle. He cites similar experiments with hirudin which were carried out by Schwalbe and by Loeb, and with peptone by Eberth and Schimmelbusch.

It is obvious, nevertheless, that slowing of the circulation is not the only factor involved in thrombus formation. Local trauma or inflammation of the vein wall, as was shown experimentally by Zahn and others, and as is seen clinically following injury or suppuration, will also lead to thrombosis at the site of vascular damage. This is the principle made use of in the artificial obliteration of varicose veins by means of injections of sclerosing substances. Intimal changes may well be a factor, too, in the spontaneous thromboses of varicose veins. However, the demonstration of gross venous lesions accompanying the bland forms of thrombosis of the larger vessels is very rare. Furthermore, the selective involvement of the left side is difficult to explain on the basis of primary pathological changes in the vein wall. In this connection, it is interesting to note that Kristenson has recently described adhesions and fold formations in the intima of a large percentage of adult iliac veins studied at autopsy. These changes are sharply localized to the left common iliac vein. He believes that they are due to pressure from the iliac artery, and considers them etiologically significant in the development of thromboses. Similar changes have been described by MacMurrich and others. Nevertheless, it still remains to

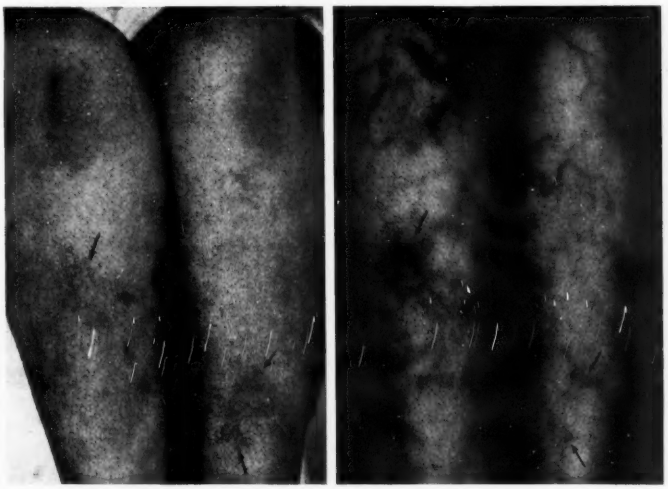

Fig. 1. Relation of varicose eczema to varicosities. Left, Patches of eczema in upper and middle portions of legs. Right, Infra-red photograph of same view, reveals varices directly beneath each patch of eczema.

be proved that such lesions of the vein wall underlie thromboses in general.

Changes in the composition of the blood which are frequently described as favoring the development of thrombosis are dehydration, increased viscosity, changes in the acid base reaction, greater fragility of the red cells, accelerated sedimentation rate, increased number and fragility of the platelets, and shortening of the coagulation time. These changes are frequently emphasized by clinical observers, but their significance has not been definitely established. The red and white corpuscles seem to play no part in the genesis of thrombosis. Changes in these structures would, therefore, seem to have little bearing on the subject. Increase in the number of platelets is often emphasized, but it is difficult to see how this finding alone could significantly determine thrombus formation. Increased friability of the platelets, alterations in their electrical charge, and greater tendency toward agglutination have also been mentioned, and might be more directly related. These are changes, however, which cannot readily be measured. Thrombosis is frequently attributed to augmentation of the clotting factors and shortening of the coagulation time. As was stated before, these elements play no rôle in the initiation of the thrombus. However, they may affect the secondary, coagulative phase, and as such, play a rôle in the propagation of the peripheral portions of the thrombus.

Phlebitis. The inflammation of the vein which invariably occurs in the conditions under discussion varies widely in its source, nature, intensity, and significance. As is seen in the accompanying tables, the range extends from simple, non-bacterial reparative inflammation following bland thrombosis, to actual suppuration.

The reparative inflammation secondary to bland and aseptic thrombosis represents the mildest grade of phlebitis. It is often so slight as to be clinically unrecognizable, and the presence of the underlying thrombosis is consequently often not recognized. Because of

TABLE I.—ORIGIN OF VEIN INFLAMMATIONS

1. Reparative reaction to bland thrombosis
2. Metastatic infections
   - A. Spontaneous phlebitis
     - a. Normal veins
     - b. Varicose veins
     - c. Buerger's disease
     - d. Phlebitis migrans
   - B. Postoperative or puerperal phlebitis
3. Extension of infection by contiguity
4. Direct injury or infection

the mildness of the reaction provoked, thrombi of this type are but loosely attached to the vein wall, and readily break loose to form emboli. As will be shown later, the sudden, massive pulmonary emboli usually arise from this type of thrombus.

In most of the phlebitides, the organisms or toxins reach the affected vein by the metastatic route. They may come in direct contact with the intima of the vessel, or may lodge in the vasa vasorum as minute infectious emboli. Aschoff is of the opinion that in this form of thrombophlebitis too, thrombosis is the primary process, and that circulating organisms are passively included in the developing thrombus. Bacterial growth within the thrombus may then lead to secondary infection of the adjacent portion of the vein wall. Metastatic infection accounts for the spontaneous thrombophlebitides of normal and varicose veins and probably for the vein inflammations of Buerger's disease and of phlebitis migrans. The postoperative and puerperal phlebitides arising at a distance from the field of infection or operation also fall within this category.

Mention should also be made of the remarkable tendency of veins to harbor latent infections for long periods of time. Repeated attacks of inflammation in the same or adjacent segments of vein are frequently due to periodic activation of the same infection, rather than to new infections. This is particularly true of the subacute and chronic forms of phlebitis involving normal and varicose superficial veins, and probably also of so called phlebitis migrans. De Takats has written of latent infections in varicose veins and Kendall and Jacques, in unpublished observations, have obtained bacterial growth from a large proportion of specimens of vein removed during ligation of the long saphenous vein in cases of extensive varicosities. The ascending phlebitis which sometimes follows the injection of varicose veins is probably due to the activation of a pre-existent latent infection.

A third possible avenue of infection to the vein wall is by extension from suppurative processes about its radicles, and progression by contiguity along the vein wall or along a thrombus forming within the vein. This type of infection may conceivably arise from an infected placental site and lead to a suppurative thrombophlebitis, by extension, of the iliac veins. A similar mechanism may explain the portal thromboses secondary to suppurative appendicitis. This method of infection, however, accounts for but a small fraction of the cases of phlebitis, and is particularly infrequent as a source of the phlebitides of the lower extremities.

A final type of phlebitis is that which arises by direct trauma to the vein wall, as from a penetrating injury or perforation by a fractured bone spicule. In the same category are direct infections of the vein arising in suppurative lesions surrounding it, and extending from the adventitia inward toward the intima. A classical example of vein infection by this mechanism is sinus thrombosis complicating mastoid suppurations. In the lower extremity, phlebitis of this type may develop secondary to infected wounds, osteomyelitis, or pelvic abscesses overlying the large veins. This group, too, comprises but a small number of the vein inflammations, and the source of the infection is usually obvious. In these there can be little doubt that the thrombosis, if it develops at all, is secondary to the intimal lesion.

Postoperative vein complications. The thromboses and phlebitides arising as complications of operation or delivery are of particularly poignant interest to the surgeon. We may, therefore, summarize those changes in the organism brought about by these procedures which are conducive to the development of vein disease. Retardation of the circulatory rate has been shown to be one of the prerequisites to the development of both bland and infective thrombi. It has long been appreciated that the rate of blood flow is retarded after operation, and Dahl has recently reported actual roentgenological demonstration of delayed venous return from the lower extremities following abdominal surgery. Slowing of the rate of blood flow after operation is occasioned by the enforced bed rest, depletion of the general condition of the patient, lowering of the blood pressure, and weakening of the cardiac impulse. Venous stagnation is further favored by inhibition of the depth of respiration because of abdominal

pain, distention, and constrictive bandaging. The blood volume is reduced as a result of hemorrhage, restricted fluid intake, and loss of body liquids through vomiting and diaphoresis.

Changes wrought in the composition of the blood by operation have been widely studied, and with varying results. Among those mentioned are increased viscosity, sedimentation rate, blood protein, fibrinogen, calcium, and platelets. Colloidal and electrical alterations, disturbances in the acid base equilibrium, and qualitative changes in the platelets have all been reported. The clotting time and bleeding time have frequently been found to be shortened. Granted that such changes may accompany the postoperative or puerperal period, it is unlikely, as has been herein shown, that most of them could have any bearing on the thrombosis question. Hypothetical alterations in the agglutinative tendency of the platelets, which might, conceivably, predispose to their settling out and becoming agglutinated, do not lend themselves to demonstration.

Alterations in the vein wall might occur as a result of operative trauma to the vessel. It is difficult to see, however, how trauma during the ordinary laparotomy, particularly for lesions in the right upper quadrant, could sufficiently irritate the veins of the left lower extremity to produce thrombosis. Even phlebitides following appendicitis operations are predominantly left sided. Furthermore, the relatively long latent period between the operation and the onset of the vein complication speaks against operative trauma as an important factor in the etiology of thrombosis.

The rôle of infection is frequently adduced in the explanation of postoperative and puerperal phlebitides, and indeed, these complications are more common in patients in whom infection is present. However, this discussion of the avenues of infection to the veins emphasized the infrequency with which direct connection between the operative or placental site and the phlebitic vessel can be demonstrated. Infections probably increase the tendency to vein complications by adding to the impairment of the general condition.

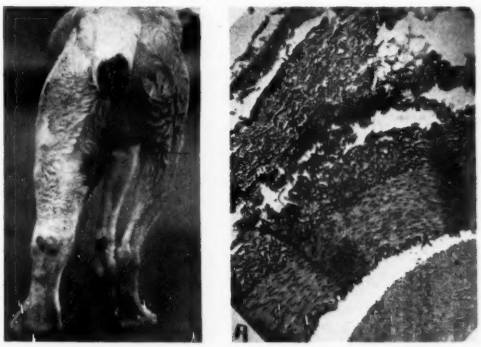

Fig. 3. Experimental thrombophlebitic edema. A, Section of artery and vein following experimental thrombophlebitis, showing absence of marked periphlebitic inflammation.

Fig. 2. Phlebitic indurations from superficial vein phlebitis without marked varicosities, in patient who has had old, deep vein phlebitis.

Fig. 4. Elephantiasis resulting from deep vein phlebitis.

prolonging the immobility and further retarding the rate of blood flow. Furthermore, infection from the field of operation may provide the atrium from which bacteria are carried to the affected vein by the metastatic route. The rôle of infection may therefore be considered as indirect and contributory rather than determinating.

Relation to embolism. Thrombosis and inflammation of the veins derive much of their significance from their ominous association with pulmonary embolism. The incidence, character, and menace of the embolism depend upon the type of vessel which harbors the thrombus, and the intensity and nature of the coexistent phlebitis. A few general rules may be stated. The firmness with which the thrombus is attached to the vein wall is apparently in direct proportion to the degree of inflammatory reaction provoked. Most of the major emboli arise from static thrombi lying loosely in the vessel. Clinically recognizable thrombophlebitis antedates such embolism only in exceptional cases. Suppurative softening, however, results in the fragmentation of the thrombus with consequent showers of small infectious emboli, which frequently lead to pulmonary suppuration or pyemia.

The size of the embolus naturally depends upon the length and lumen of the vein in which the antecedent thrombus is lodged. Massive emboli, striking without warning and frequently bringing sudden and dramatic death, must be large enough to obstruct one or more of the main branches of the pulmonary artery. Such a mass can come only from a major vessel, usually the iliac or femoral vein. Small emboli frequently result in pulmonary infarction, particularly if there is coexistent respiratory or cardiac impairment. Many, of smaller size, produce no detectable clinical disturbances. The infrequency of infection in infarcted lung has been offered as evidence of the non-bacterial origin of the thrombus.

PHLEBITIS AND THROMBOPHLEBITIS OF THE LOWER EXTREMITIES

Thrombosis and thrombophlebitis are especially likely to occur in the veins of the lower extremities. The clinical manifestations and the gravity of the disease depend, in large part, upon the type of vein affected. Several years ago, Homans suggested dividing the phlebitides of the lower extremities into three groups: those affecting the deep veins, those of normal superficial veins, and those involving varicose veins. This convenient classification will be followed in this discussion (see Table II).

TABLE II.—CLASSIFICATION OF PHLEBITIDES OF LOWER EXTREMITIES

1. Phlebitis of varicose veins
   - A. Ascending type
   - B. Localized form
2. Phlebitis of non-varicose superficial veins
   - A. Ascending
   - B. Localized
   - C. Phlebitis migrans
   - D. Phlebitis of Buerger's disease
3. Deep vein thromboses and inflammations
   - A. Suppurative thrombophlebitis of iliac veins
   - B. Non-suppurative thrombophlebitis—phlegmasia alba dolens
   - C. Bland thromboses

Phlebitis of varicose veins. Thrombosis and inflammation are extremely common in varicose veins, and most of the complications of varicose veins may be shown to be inflammatory in origin. In light of what has been said regarding the etiology of phlebitis, it is not surprising that these veins which are subjected to stasis and its attendant pressure and anoxemia, provide a site of predilection for circulating organisms. Frequent degenerative changes in the lining endothelium further favor the development of thrombosis and inflammation. Conditions predisposing to thrombosis in general, such as operation, delivery, intercurrent infection, debility, and trauma are frequently the immediate precursors of the phlebitis, although repeated attacks of spontaneous inflammation are extremely common in patients who consider themselves otherwise entirely well.

Homans called attention to the relative mildness of the clinical manifestations of varicose vein phlebitis as compared with that in normal vessels. He states further that the sequelæ of these inflammatory attacks are of little significance. This observation is not borne out by the study of large numbers of these patients. It can be shown rather convincingly that most of the complications of varicose veins, which are usually attributed to stasis and congestion, are really inflammatory

in origin, and that there is a direct sequence of changes from the phlebitides to indurations, dermatoses, and ulcerations.

The attack of phlebitis may follow a slight bruise or come in the train of an acute upper respiratory infection. Often no ascribable cause can be found. Two rather distinct varieties are seen. The inflamed segment may involve but 2 or 3 inches of the vein and remain localized to this area, or it may advance centrally and peripherally until most of the saphenous system is involved. Mild general symptoms may accompany the latter; in the former, they are usually absent. The patient complains of slight pain in the involved area, and, on examination, a firm, dull red, somewhat tender cord is seen. A variable amount of periphlebitis accompanies the attack. These inflammations pursue a subacute course, and recurrences in the same or other segments, are notoriously frequent.

In the progressing form of the disease, the inflammation almost invariably stops short at the saphenofemoral junction. Limitation of the process to the superficial veins has been convincingly explained on the basis of the difference in rate of blood flow in the superficial and deep venous systems. The circulation in the latter is sufficiently more rapid to prevent the extension of the phlebitis to the femoral vessels. The thrombus almost invariably canalizes so that the varicosity is rarely permanently obliterated by this process. Calcification of portions of the thrombus occasionally occurs, with formation of phleboliths which persist indefinitely. In rare cases, suppuration of portions of the thrombus may result in abscess which requires drainage.

The usual type of varicophlebitis remains localized, with greater tendency to recur at the same site. Eventually, the inflammatory changes extend from the vein wall to the adjacent skin and subcutaneous tissues. At this stage the lesion presents the appearance of a round or oval patch of low grade cellulitis. The skin is reddened and indurated, and there is slight pain and tenderness on pressure. The temperature may be elevated a fraction of a degree. These attacks, too, tend to recur, and as they recede, leave pigmentation and fibrosis. Ultimately, the skin over a limited area, which sometimes involves a large portion of the circumference of the lower leg, becomes transformed into a dark brown, indurated zone with pigmentation, fibrosis, and atrophy of the overlying integument. These zones, which are spoken of as phlebitic patches or phlebitic indurations are the immediate precursors of varicose eczemas or of ulcers which show little tendency to heal.

That these changes are inflammatory, rather than merely stasic, can be convincingly demonstrated by following the lesion through its successive stages. The long axis of the patch is usually parallel with the course of the infected vessel, and in many instances, a varix may be palpated as a groove or channel passing through the board-like area. There is no parallel between the extensiveness of the varicosities and the severity of these skin lesions, nor does the area of involvement always lie in the most dependent portions where congestion would be most severe. Adjacent portions of the skin, separated from these indurations by only a few millimeters, retain their normal color and consistency. It is difficult to reconcile the sharp limitation of the process with chronic passive congestion, in view of the very rich overlapping of the cutaneous venous circulation. Finally, in support of the inflammatory genesis of these complications, it may be mentioned that conditions of known chronic passive congestion, such as occur in long standing cardiac decompensations, are not associated with indurations of the type described here, or their subsequent degenerations.

Aside from these local changes, which may become disabling, the prognosis in varicophlebitis is good. Embolism, the dreaded sequel to any phlebitis, is very rare. This is particularly true of the artificial, or chemical phlebitis induced by the therapeutic injection of varicose veins. Two factors of safety are mentioned in explanation of the infrequency of embolic accident following such injections, and these apply largely to the spontaneous phlebitides as well. The more important of these is the fact that the thrombus is inflammatory in its origin and is firmly adherent to the irritated and denuded vein intima. Such thrombi are not at all likely to become dis-

lodged as emboli. The second factor is the reversal of the direction of flow in varicose veins, as may be demonstrated by the Trendelenburg test. If portions of the clot did break loose while the patient was erect, they could not flow upward to enter the general circulation, but would, of necessity, go downward, to be caught in the pre-capillary vessels, where they could do no harm. The rare emboli which do occur, are of small size, and give rise to minor pulmonary infarctions. If infected, suppuration might result.

Treatment. Inasmuch as the sequelæ of varico-phlebitis frequently lead to distressing and even disabling end-results, their prevention constitutes the greatest indication for the early obliteration of varicose veins. Injection or ligation in the presence of active phlebitis, even of the subacute variety, is dangerous, and such treatment should be postpoined for several weeks or months after the inflammatory phenomena have disappeared.

Active treatment for this type of phlebitis is essentially conservative. Prolonged immobilization is unnecessary, and most of these patients may be permitted to be ambulant throughout. If the involved vein segment is below the knee, the limb should be encased in an Unna's paste boot, which is renewed periodically. This relieves the edema and congestion, and materially alleviates the discomfort. When the inflammation has subsided, further support by means of woven elastic bandages, for an additional period of several months is advisable. The progressing type of phlebitis involving the saphenous trunk in the thigh is not amenable to this simple therapy, and rest, elevation, and the application of hot compresses may be required. The progress of the lesion may sometimes be checked by compressing the trunk of the vein above the highest point of involvement by means of a felt pad held in place with a strip of adhesive tape. Obliteration of the trunk in advance of the lesion by injection or ligation has been advised by some writers, and, in my opinion, may be indicated in cases in which showers of small emboli are being thrown off. This latter eventuality rarely occurs. After an adequate period of quiescence, cautious treatment for the underlying varicosities should be instituted. If the first small, tentative injections produce no excessive inflammatory response, active obliteration by means of injection, together with ligation, if necessary, should be carried out.

Phlebitis of non-varicose superficial veins. Superficial veins of the lower extremities which are not varicose are also subject to phlebitic inflammation. Such normal vessels are much less frequently involved, however, than are those previously dilated and varicose. The difference in incidence may readily be explained by the relative absence of stasis and of degenerative intimal changes in the intact veins. The clinical course and the sequelæ of phlebitis of these veins closely resemble those of inflamed varicosities. Homans stresses the benignity of phlebitides of varicose veins as compared with normal superficial vein inflammations, and explains the difference on the basis of gradual adaptation of the lymphatics during the period of stretching and dilatation of the veins, so that when the eventual phlebitic lymphangitis occurs, no crippling of the lymphatic return takes place. Our experimental observations, which will be discussed later, and my clinical experience is at variance with this view. Edema of the extremity is not part of the picture of superficial vein pathology, whether those superficial veins be normal or varicose, and the local cutaneous changes are equally severe in both types of phlebitis. As was stated before, they rest upon direct inflammatory changes emanating from the inflamed vein segment, and are not directly due to stasis in either the venous or lymphatic channels.

Phlebitis of normal superficial veins may be spontaneous or may result from local infections in the vicinity of the affected vessel. I have seen progressive thrombophlebitis of the entire saphenous vein following a localized third-degree burn on the dorsum of the foot. These inflammations are also prone to occur after operation, childbirth, or trauma, and may complicate infectious diseases or cachectic states. They are particularly common in limbs which have been the seat of deep vein phlebitis, many of the distressing and disabling sequelæ of which may be due to associated or secondary superficial vein infection.

The clinical manifestations of this type of phlebitis are similar, as stated, to those described of the varicose vessels, although they may be somewhat more acute. The same two rather distinct varieties are seen. The progressive or advancing type commonly occurs after operation or delivery. It usually involves the trunk of the long saphenous vein and frequently begins in the upper portion of the thigh. It is ushered in with fever and pain in the limb, and a firm, tender, reddened cord may be detected along the course of the saphenous vein. General edema of the leg does not develop. The process may progress downward to involve the entire saphenous system and occasionally may extend to the communicating veins which anastomose the superficial with the deep systems. When the process subsides, the thrombus canalizes and the lumen of the vein is restored. The wall is somewhat thickened and the valves within the diseased segment, as a rule, are completely destroyed. Homans has pointed out that although the circumference of these vessels may not be materially increased, the absence of functioning valves permits a much greater degree of regurgitation than occurs in extensively dilated and tortuous varicose veins. In the exceptional case in which the valves in the communicating as well as the saphenous veins are crippled, reflux takes place through both channels, with very serious derangement of the venous return from the skin areas of the leg.

The localized form of superficial vein phlebitis usually occurs in the calf or lower half of the leg, frequently spontaneously or after slight trauma. It is more common in women, usually in those of middle age, and resembles in its clinical manifestations and sequelæ, the ordinary form of localized phlebitis of varicose veins. Many so called varicose ulcers arising in the absence of demonstrable varicose veins are due to this type of phlebitis. It has a special predilection for limbs which have harbored deep vein infections, and leads to indurations, dermatoses, and intractable ulcerations, all of which materially aggravate the distress and disability of this unfortunate group of patients.

Major embolism does not complicate superficial vein phlebitis. Repeated small infarcts may result from dislodged fragments of thrombus from the advancing type of phlebitis; they are rare in the localized form. Treatment of both forms is similar to treatment of the corresponding types in varicose veins. Rest, elevation, and heat usually suffice for the progressive form. Showers of emboli may require ligation. When the process has entirely subsided, the resulting incompetent vein may require obliteration preceded, perhaps, by ligation. Supportive treatment with the Unna boot materially alleviates the inflammation, and also relieves the pain and itching and promotes the healing of the resulting eczemas and ulcers. These phlebitides are notoriously prone to recur, each succeeding attack increasing the induration and discoloration, and adding to the permanent, irreversible impairment of the tissues. Constant supervision is therefore necessary, with prompt treatment at the first sign of recurring trouble. In the intervals between attacks, measures directed toward improving the circulation, including contrast baths and diathermy, may be indicated.

Phlebitis migrans and the phlebitis of thromboangiitis obliterans. A form of superficial vein thrombophlebitis has been singled out as an entity under the name of phlebitis migrans. This rather uncommon disease affects males predominantly, usually those of youthful or middle age. It is characterized by recurring attacks of low grade inflammation of the superficial veins, usually involving localized segments of the saphenous trunk or its tributaries, and frequently displaying a roughly symmetrical distribution. Its course is marked typically, by periodic subsidence and recurrence in the same or other segments of superficial veins. Extension to the deep vessels, and involvement of visceral veins in the chest and abdomen have been reported. There are no specific pathological or bacteriological findings.

Lesions of this type have been described by Paget, as manifestations of gout. Similar affections have been attributed to syphilis or have been observed as complications of rheumatic fever and other diseases. Buerger has called attention to the frequent occurrence of patches of superficial vein phlebitis, resembling those here described, in patients with

thrombo-angiitis obliterans. This finding has been widely confirmed, and recurring superficial phlebitis is now considered highly suggestive of Buerger's disease. This raises the suspicion that many of the so called "idiopathic" and gouty forms of phlebitis migrans may actually have been early manifestations of thrombo-angiitis obliterans. Cases have, in fact, been reported by D'Abreu and others, in which Buerger's disease was recognized as long as 40 years after the onset of the migrating thromboses. Since recurrence is a characteristic feature of superficial vein phlebitis in general, and in view of the absence of pathognomonic morphological and bacteriological findings, and inasmuch as many of these lesions are part of the picture of Buerger's disease, the justifiability of considering phlebitis migrans a clinical entity may be questioned.

PHLEBITIS AND THROMBOSIS OF THE DEEP VEINS

By far the most important group of vein occlusions and inflammations, from the standpoint of severity of their clinical manifestations, sequelæ, and danger to life, are those involving the deep veins. These vary in degree of inflammatory reaction from the suppurative thrombophlebitides secondary to purulent postpartum or postabortive pelvic infections, through the ordinary deep vein phlebitides as they occur after operation or delivery—the so called phlegmasia alba dolens—to the bland thromboses of the iliac and femoral veins whose existence is unsuspected until a massive and perhaps fatal pulmonary embolism supervenes. Some degree of inflammatory reaction, as has been emphasized before, is always present, whether it be the primary factor, a secondary phenomenon, or merely the reparative reaction to a simple static thrombosis.

Suppurative phlebitides extending into the iliac vessels from purulent infections in the pelvis, by way of the uterine veins, are characterized essentially by septic phenomena which may lead to multiple abscesses in the pelvis, or to fatal septicemia or pyemia before local signs of vein occlusion appear. Repeated chills, high remittent fever, intense toxemia, and ileus dominate the picture. Circulatory disturbances in the lower extremity, such as cyanosis or edema, are not observed. Massive embolism is infrequent. Showers of small septic emboli often give rise to septic infarcts or pneumonias, or even to generalized metastatic pyemic abscesses. The prognosis in this type of phlebitis is always grave, and heroic therapeutic measures, such as the intra-abdominal ligation of the iliac veins or the intravenous injection of antiseptic dye solutions may reasonably be considered.

The usual non-suppurative postoperative or postpartum thrombophlebitis, long recognized under the name of milk leg or phlegmasia alba dolens, presents a characteristic picture. The lesion may develop at any age, and is more frequently seen after septic operations or deliveries. The onset of the vein infection is frequently announced by a chill, which may be repeated, and a rapid rise in temperature. The patient complains of pain in the leg or thigh, stiffness, and loss of motion. The limb begins to swell, the edema usually appearing first in the lowermost portions of the leg. Its upward extent depends upon the degree of venous occlusion. The leg becomes uniformly swollen, tense, white and shiny, and pits on pressure. There may be tenderness along the course of the deep vessels. Fever persists for days or weeks, and after subsidence, may recur. The edema disappears in the milder cases, to recur for a short time when the patient begins to be up, and may then vanish entirely. In more severe involvements, the edema persists for a number of months and in some cases is permanent. These familiar porky edemas may be a source of severe discomfort and disability, especially if repeated attacks of an erysipelas-like streptococcic cellulitis supervene. The skin then becomes thickened and infiltrated, and a true elephantiasis may develop. Late superficial vein inflammations, with their sequelæ of indurations, dermatoses, and ulcers are frequently superimposed upon the picture, as was mentioned before, thereby adding materially to the distress and disability of these unfortunate sufferers.

The cause of the edema which follows thrombophlebitis has long been disputed. Two principal explanations have been offered,

one ascribing the edema to interference with the venous return, the other attributing it to inclusion of the main lymphatic channels in the perivenous inflammatory reaction, resulting in lymph stasis which is considered the source of the edema. We have shown (31, 32, 33, 34) that mechanical interruption of the lymphatics of the limb of the experimental animal does not result in edema. Extensive peripheral obstruction of the main veins, however, by means of even the blandest thrombi, uniformly results in massive edematous swelling of the extremity. India ink emulsion injected into the feet of animals at the height of experimental edema can be recovered from the iliac lymph nodes, indicating patency of the lymphatics. This, together with the absence of microscopic evidences of perivenous inflammation in these experiments, would make it seem that the lymphatic theory of postphlebitic edema is not tenable, and that the swelling is to be explained on the basis of insufficient venous return.

A second factor which may add to the swelling of the leg is the collateral inflammatory edema which accompanies the infected portions of the vein. Some authors limit the usage of the term "phlegmasia alba dolens" to those cases in which the edema is inflammatory rather than circulatory in origin. The former is supposed to begin in the upper portions of the thigh, and extend downward with the advance of the phlebitis. The latter is said to appear first in the most dependent portions, its upward extent depending upon the degree of circulatory embarrassment. It seems to me the distinction is difficult and unnecessary, nor was it made when the term was first applied (29). These cases are better considered as a single group, and the term "milk leg" or "phlegmasia alba dolens," if used at all, should cover all cases of postoperative or postpartum thrombophlebitis with edema.

Thrombophlebitis of this type is often feared as the source of fatal pulmonary emboli. While embolism does occur, it is relatively infrequent, and more often than otherwise, takes the form of pulmonary infarction which causes little or no clinical disturbance, or which may simulate pneumonia. The relative infrequency of embolic accident is explained by the material degree of inflammation which accompanies and fixes the thrombus. Furthermore, the veins in these cases are completely occluded, as is evidenced by the dependent edema. A thrombus in such a vessel is much less likely to break loose than is one lying within a vein through which the blood is still flowing.

The status of the venous return from the extremity after recovery from a deep vein thrombophlebitis has received but little attention in the literature. In some cases, the edema disappears entirely. In many others, its persistence for indefinite periods indicates, among other things, that the venous drainage has been permanently damaged. Actual, persisting occlusion of the principal deep veins is extremely uncommon, in spite of the usual textbook warnings to rule out deep vein closures before destroying superficial veins. In an extensive material, over a number of years, I have seen but two instances in which I was convinced that the deep veins remained impatent. In the vast majority of patients who have had deep vein thrombophlebitis, evidences may be found to show that the lumen of the veins has been restored. If superficial varicosities are present, the Trendelenburg and Perthes tests almost invariably point to patency of the deep veins. The edema usually subsides, at least in part, with recumbency or elevation of the extremity, and recurs when the patient is up and about. These findings indicate that canalization of the thrombus almost always follows deep vein inflammations. The restoration of the lumen, however, is accompanied by a destruction of the valves, resulting in a relative functional insufficiency of the venous return.

In our studies of thrombophlebitic edema, the protein content of the edema fluid was found to be relatively high, values between 3 and 4 per cent often being recorded. Of the multiplicity of factors involved in maintaining the water balance between the capillaries and the tissue spaces, the colloid osmotic tension exerted by the plasma proteins of the blood is recognized as one of the important elements in the return of fluid from the tissue spaces to the blood vessels. The extravasation of

edema fluid containing a high concentration of these same proteins tends to equalize this colloid osmotic tension and interferes with the fluid return. In later stages, there are probably irreversible changes in the tissues, of a physical nature, which further tend toward perpetuating the edema. It would, therefore, seem therapeutically important to hasten, as much as possible, the resorption of the edema in the acute stage. We have demonstrated, in our experimental studies, that the disappearance of this type of edema may be accelerated by the intravenous use of mercurial diuretics such as salyrgan, by the parenteral administration of calcium salts and parathyroid hormone, and by X-ray therapy. These features have been included in our management of acute thrombophlebitides with edema.

Treatment during the acute stages of iliac and femoral thrombophlebitis is directed toward the prevention of embolic accident, the acceleration of the resorption of the accompanying leg edema, and symptomatic relief for the associated discomfort. The former requires rigid bed rest, elevation, and the avoidance of all massage, manipulation, and rough handling which might dislodge a portion of the thrombus. These precautions are observed in spite of the fact that massive embolism is an uncommon complication in this group of vein lesions. The duration of the enforced rest is still a perplexing question. Varying periods, ranging from 20 days to 6 weeks after the temperature has become normal, have been arbitrarily established by various writers. The tendency of late has been to shorten this time. In a recent publication, de Takats has described a management of cases of this type, in which gradual exercise and massage are begun after 10 days of normal temperature, which allows the patient out of bed during the fourth week.

Early mobilization of the edema is effected by elevation, restriction of the salt and fluid intake, and administration of salyrgan intravenously in doses of 1 to 2 cubic centimeters. Ammonium chloride may also be given. Halban has advocated X-ray therapy during the acute stages of the disease. Our experiments revealed that X-ray irradiation accelerated the rate of fluid absorption in postphlebitic edema. In a few cases treated with X-ray, rapid subsidence was noted. Calcium and parathyroid therapy has long been empirically employed to hasten absorption of exudates. Experimentally, too, this regimen was found to hasten the disappearance of the edematous swelling. As soon as the subsidence of the inflammation permits, graded exercise, cautious massage, and diathermic treatments are instituted. Support by means of elastic bandage or stocking is advised for a period of 3 to 6 months, and longer, if the edema persists.

In the chronic stages of phlebitic edema, particularly in those associated with indurations, ulcerations or elephantiastic changes, treatment is difficult and unsatisfactory. The Unna's paste boot offers an almost ideal means of obtaining elastic support, whereby the edema may be reduced, the indurations softened, and the ulcers healed. When maximal improvement has been attained, further supportive treatment with elastic bandages or stockings is advised, and massage and diathermy are employed. Constant supervision is necessary, in order that recurrent inflammations may be recognized and controlled before extensive ulcers again develop.

Surgical procedures such as the Kondoleon operation, which are directed toward the establishment of additional communications between the superficial and deep lymphatics of the leg are frequently advised for the elephantiasis which may complicate postphlebitic states. As was shown by our experimental investigations, the hypothesis upon which these operations are based is untenable, because the lesion is one of the veins rather than of the lymphatics. Furthermore, the primary site of inflammation is generally considered to be in the iliac and femoral vessels. Both superficial and deep lymphatics drain into the trunks surrounding these veins. It is difficult to see how anastomosing the lymphatics, even if it were possible, distal to the site of obstruction, could affect the lymph drainage of the extremity. Nevertheless, experience in our own and other clinics attests the value of the Kondoleon procedure in cases of this type. The improvement resulting can probably be explained on the basis of the excision of irreversibly altered skin and sub-

cutaneous tissue, rather than upon the improvement of the lymphatic circulation.

The bland thromboses of the main veins of the lower extremities differ sufficiently in their clinical manifestations from those already described to warrant separate consideration. This is especially true because of their sinister relation to pulmonary embolism. From many sources there have been statistical reports showing an appalling increase in the frequency of thrombosis and embolism. This is particularly true of the German literature, and, to a lesser extent, of the French and British reports, and applies to medical as well as postoperative material. According to Rosenthal, a similar increase has not been observed in the United States. Many factors have been blamed for the increase in thrombosis, including the impaired post-war nutritional states, the increase in intravenous medication, and the broadening of surgical indications to include many patients of advanced age, in late stages of disease, and with impaired hearts and blood vessels, who offer greater predisposition to vein complications. Because of the importance of thrombosis and embolism as a cause of postoperative death, the study of all possible predisposing influences and their eradication is of the greatest importance.

As a result of their insidious onset and the absence of striking clinical signs to indicate their existence, a sudden fatal embolic accident during convalescence from an otherwise uncomplicated operation is usually the first intimation that a thrombosis has occurred. Examination of the case records in retrospect often reveals a subfebrile elevation of the temperature for several days before the embolism occurred, which might have given an intimation that a thrombus had formed. A step-like rise in pulse rate has also been described as a diagnostic sign which, however, has been found lacking as often as it was present. Ducuing and his collaborators have closely studied the symptoms of simple thrombosis, and have described in detail various types of pain, spontaneous and induced by pressure or exertion, which may permit the early diagnosis of thrombosis and appropriate prophylactic immobilization. They list three sites of predilection for this pain: in the foot, the calf, and the thigh, and believe that careful search and attention to these symptoms have permitted diagnosis of thrombosis in a far larger percentage of cases than is usual, and that the resulting immobilization has materially reduced the incidence of embolic accident.

The prognosis in bland thrombosis is always grave because of the ominous tendency of these static, non-adherent clots to become detached as emboli. Furthermore, the lesion characteristically involves the large veins of the pelvis and lower extremity, so that emboli which do break loose are often sufficiently large to occlude the main branches of the pulmonary artery and lead to immediate or early death of the patient.

Treatment for thromboses must necessarily be largely prophylactic, since the presence of the condition is usually unsuspected. The factors responsible for thrombosis have been enumerated. The most important of these, slowing of the circulation, is the one most readily avoided. Efforts to combat the lowered blood pressure and slowed circulation have consisted in the avoidance of constricting bandages, the inhalation of carbon dioxide to promote deeper respiration, the administration of thyroid extract, the use of systematic exercises beginning immediately after operation, and the early mobilization of surgical patients. Experience in getting patients out of bed within the first 2 or 3 days after laparotomy has accumulated in various clinics over a period of years. It has been found to be safe, beneficial to the general physical and mental well-being of the patient, and effective in reducing the incidence of thrombosis and embolism. In patients too debilitated to permit mobilization, systematic bed exercises afford the best substitute. These should find introduction in the postoperative management of all patients after major surgical procedures.

Active treatment for thrombosis presupposes its diagnosis before embolism has occurred. If it is at all suspected because of subfebrile temperature elevation, tachycardia or pain in the foot or leg, the greatest possible caution to prevent dislodging emboli would be called for. This would entail the gentlest

possible handling during nursing care, particularly during the bath and in the use of the bed-pan. Because the presence of the thrombosis can only be suspected, the efficacy of any management cannot be clearly evaluated.

A few patients have been rescued from embolic death by successfully executed Trendelenburg embolectomy. Credit is due the surgeons who have saved lives by this heroic measure. The difficulty of being on hand when the accident occurs, the uncertainty of diagnosis, the severity of the intervention, and the haunting question as to whether the patient might not have survived if not operated upon will probably keep the Trendelenburg operation a rare occurrence, and the patients saved by it exceptional indeed. In prophylaxis rather than in therapy is our hope.

SUMMARY

1. Phlebitis, thrombosis, and thrombophlebitis are considered under a single heading because the three terms are frequently interchanged, and because there is no clear-cut differentiation between them. Phlebitis is usually accompanied by thrombosis and even the blandest thrombosis provokes an inflammatory reaction of the vein wall. It is often impossible to determine whether it is the thrombosis or the phlebitis which is the primary condition.

2. Thrombosis consists of the agglutination of platelets along the vessel wall to form a homogeneous mass. Superimposed upon this is a deposition of white blood corpuscles. Fibrin formation plays no part in the origin of the thrombus, but may occur at its periphery as a result of the liberation of ferments from the agglutinated platelets and corpuscles, which results in the coagulation of the adjacent blood mass.

3. Thrombosis is due to the interaction of two or more of the following causes: Slowing of the circulation, which is probably the most important factor; changes in the vessel wall, which is the initiating factor in some types, but of secondary importance in others; and alterations in the composition of the blood. The latter are probably more important in the propagation of a thrombus already formed, than in its origin.

4. Phlebitis may be merely the reparative reaction to a bland thrombosis; may be due to metastatic infection, may reach the vein by extension from a suppurative process about the radicles of the vessel, or may result from direct injury or infection. Veins have a remarkable capacity for harboring latent infections, and periodic activation frequently occurs.

5. Vein complications frequently follow delivery or operation. The changes induced in the organism by these procedures consist of slowing of the circulation and, perhaps, as yet undemonstrable blood changes. Infection indirectly favors the development of vein disease by providing an atrium for infecting organisms and by further slowing the circulatory rate.

6. The danger of embolism is inversely proportional to the amount of inflammation accompanying a thrombus. Suppurative softening may lead to showers of small infective emboli. Massive and perhaps fatal emboli can arise only from major vessels, usually iliac or femoral veins. Smaller emboli may produce little or no clinical manifestations.

7. Phlebitis is the most common complication of varicose veins, and a direct sequence of inflammatory changes leads from the phlebitis to ulcers and eczemas. Two forms are described. The clinical manifestations are mild. Treatment consists in supportive therapy followed by obliteration of the veins when the phlebitis has subsided.

8. Phlebitis of non-varicose, superficial veins occurs much less frequently than varicose vein phlebitis. The two conditions resemble one another closely in their types, clinical manifestations, and sequelæ. Non-varicose vein phlebitis is particularly frequent in patients who have had deep vein infections, and materially aggravates the distress and incapacity thereby produced.

9. Deep vein phlebitides are divided into three forms: suppurative thrombophlebitis secondary to purulent pelvic infections is frequently rapidly fatal; non-suppurative thrombophlebitis, phlegmasia alba dolens, produces a characteristic picture; edema, transitory or permanent, forms an essential feature of this picture. Bland thrombosis is

usually not recognized until a massive pulmonary embolism has occurred. Treatment is therefore of little avail, and prophylaxis is of predominant importance.

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CONSERVATIVE AND RADICAL MEASURES IN THE TREATMENT OF ULCER OF THE LEG[1]

A Study of Techniques, Indications, and Results

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THE increased incidence of chronic leg ulcer in this and in other countries caused by modern living conditions has lately stimulated widespread interest. The difficulty of accomplishing cure has been a great challenge of recent years for a more adequate study of the problem. The manner in which this challenge has been met and in which treatment has been put on a more rational basis will be described in this report and a second one which will follow.

Until lately treatment has been needlessly complicated and expensive while results have generally been unsatisfactory. Referring to England, A. Dickson Wright says:

> No one realizes what a terrible curse varicose ulcer is to the lower classes of this country; it is much more prevalent than is imagined, because most of the sufferers, tired of receiving no relief from the medical profession, settle down to endure their complaint with occasional extravagances in the form of quack remedies. In every large town of the midlands may be found an institution, under the supervision of unqualified people, for the treatment of "bad legs."

It is equally true that varicose ulcer is very prevalent in this country. A patient with an ulcer of the leg is at a great disadvantage, but the unfortunate individual with a large indolent one as a rule leads a very unhappy life and invariably is almost a total economic loss. One apparently healthy man of 40 who recently came to the Vanderbilt University Dispensary had been out of work for a year and a half because of two extensive leg ulcers which, with the usual medical attention and home care, had refused to heal. As far as his incapacity was concerned he might as well have been suffering from advanced pulmonary tuberculosis. The problem was made more difficult by the man's desire to work when he was really unable to do so. Such patients as this one are seen in private practice rather infrequently but in our experience they are extremely abundant in the surgical dispensaries of this country. At Vanderbilt University Hospital in the past four and a half years 234 patients presented themselves for treatment of chronic leg ulcer. During 1933, 83 such patients were treated—an increase of 35 over the previous year.

Only within the last few years have any reliable techniques been worked out for the cure of leg ulcer. These developments have come about along two different lines of approach: the one employing radical or operative measures, the other conservative or non-operative. Both require, as a preliminary procedure, the treatment of any underlying or predisposing cause of ulceration such as varicose veins or syphilis.

Radical treatment by excision and skin grafting will yield excellent results in the vast majority of cases. Recently we have described our technique employing a full thickness graft of a modified type—the perforated or "sieve" graft, and have reported a series of cases (2, 3) cured by this procedure.

With the establishment of precise and clear cut indications for the use of these two techniques—the radical and the conservative—we have found that almost all non-malignant ulcers of the leg may be cured. Since we have already described the radical or operative technique elsewhere, we will proceed with a description of developments in conservative methods and will finally state our conclusions as to the indications for each.

CONSERVATIVE TREATMENT

The chief contribution of recent years in conservative measures has been the development of the elastic adhesive plaster technique. This development has undoubtedly been a gradual evolution brought about by efforts toward simplification in the treatment of leg ulcers. It appears to be an effective combination of four methods, each of which has been previously employed in the treatment of this

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condition. It combines in one bandage the protective and supportive qualities of an Unna's paste boot, the elastic qualities of a rubber stocking, the porous qualities of a gauze dressing, the non-slipping fixative qualities of adhesive.

MATERIAL

The elastic adhesive bandage in its usual form consists of a thin adhesive plaster mass coated on a cloth bandage. The cloth is known as domette and is so woven as to be quite elastic. It may be stretched lengthwise to about one fourth again its length and regain its original size when it is released. The width remains practically unaltered when the bandage is stretched. The adhesive mass is also elastic. While the exact proportions of its ingredients have not been made public, according to one manufacturer it contains para-rubber, rosin, organic fat, amylum, and approximately 30 per cent zinc oxide. Another states: "There is being used pure zinc oxide in conjunction with pure para-rubber and some medication," and adds that the plaster mass is produced in such a way that its elasticity is equal to that of the elasticity of the bandage. The loose weave of the cloth and the fact that the adhesive coating is thin, allow the bandage to be sufficiently porous to permit fairly free discharge to occur from an ulcer. Since the bandage is practically waterproof exudate may be washed from its external surface without disturbing it. We have used two preparations with good results. These are an imported German plaster "lomaplast,"[1] and a second called "elastoplast"[2] manufactured in this country. A third is also on the market, viz. an English preparation prepared by the formula of A. Dickson Wright and known as "flexoplast."[1]

Most of our studies have been made while using the "lomaplast" but recently this has been difficult to obtain and we have substituted the "elastoplast." The "lomaplast" seems to require greater force to stretch it than the "elastoplast," and this seems to us to be an advantage although we have no absolute proof of this point.

[1] May be obtained from G. Kuhlmann, 62 W. 45th Street, New York City.

[2] May be obtained from The Duke Laboratories, Inc., Long Island City, New York.

We believe that Wright is responsible for first pointing out the advantages of a bandage of this type in promoting the healing of varicose leg ulcers. In May, 1930 (5) and again in December, 1930, Wright (6) emphasizes the unusual results which he had obtained with this dressing. The next year (7) he published a report with a grand total of "525 cases of cured ulcer of a total area of a little over twenty-five square feet." The largest of these, 180 square inches (or 1¼ square feet), healed as readily as any. In this series there were relapses in only 3.4 per cent in 3 years, and all yielded easily to treatment. These results are especially striking when one considers how many patients with varicose ulcer ordinarily drift from place to place.

FIELD OF USEFULNESS

Granted that varicose leg ulcers form the commonest class of cases amenable to treatment by elastic adhesive they are still only one of many classes of cases in which it is useful. I have used it very successfully in promoting healing in many other types of leg ulcer such as traumatic, syphilitic, and trophic, as well as in ulcers from burns, and decubitus, and open wounds resulting from the operative removal of skin for grafting (Figs. 1 and 2). In short it forms an excellent protective and semi-occlusive strapping for any superficial wound which is not acutely infected and in which there is so much loss of skin that suturing is impracticable.

Since elastic pressure is needed for varicose veins it is especially useful as a spiral bandage in covering both veins and ulcers in the lower extremities but I have also used it as a simple covering for ulcers on the upper extremities and for wounds on other parts of the body. Its use in clean wounds from burns and in skin grafted areas will be described in a separate article now in preparation.

TECHNIQUE OF TREATMENT

Treatment of underlying causes of ulceration. In every case the cause underlying the ulcer formation should be determined if possible and remedied either before or during the local treatment with elastic adhesive. General measures should always include a thorough

physical and laboratory examination. Many patients with ulcers suffer from secondary anemia. This should be remedied and a properly balanced diet including the necessary vitamins should be prescribed. The general condition of these patients is a very important factor in their healing. Syphilis is a not infrequent cause of leg ulcer. When present, treatment will aid materially in the rapidity of healing. Granuloma inguinale and tuberculous ulcers of the skin will each need appropriate general measures until the specific infectious agent is eradicated. In chronic osteomyelitis dead bone must be removed and the cavity must be allowed to fill up by granulation before the remaining ulcer is strapped. Periostitis is usually secondary to ulceration, and will not require any especial treatment. If, however, bony spurs form, as may frequently happen in the region of the foot or ankle, these should be excised. It goes without saying that in decubitus the local cause should be removed before treatment of the ulcer. When elephantiasis exists its treatment, if operative, should precede that of the ulcer. A very persistent multiple ulceration of the legs due to infection by Staphylococcus aureus is rather common in children. Autogenous vaccine during treatment has proved of value in about half of these cases. Lastly to this admittedly incomplete list, we add varicose veins. Very few ulcers will heal without recurrence unless varicose veins are remedied. Many will heal quickly when this is done. Newell and others have pointed out the superiority of the sclerosing treatment to operation in the vast majority of cases. It is the only means of satisfactorily treating veins in ambulatory cases. We believe, nevertheless, that some veins high up in the thigh should be stripped and the saphenous ligated in order to avoid recurrence both of the veins and of the ulcers.

This list of predisposing causes while not complete may serve to emphasize the necessity of a careful diagnosis and treatment of underlying causes of ulceration before making an attack upon the ulcer.

Preliminary local treatment of ulcer. In case the ulcer area is suppurating only moderately this step may be omitted. In fact some authors advise beginning the elastic strapping immediately, no matter how soiled the area. In cases in which there is marked suppuration it is our custom over the ulcer and surrounding skin to use packs of gauze covered with cellophane or oiled silk and soaked with a saturated solution of boric acid. The latter may be introduced under the edge of, or through a small opening in, the cellophane, and renewed at 4 hour intervals. A warm water bag is used for heat over the dressing. These packs are changed at intervals of 24 to 48 hours and are kept on until the granulations are healthy in appearance and suppurating only slightly. The ulcer is then considered ready for the third step in the treatment.

The technique of applying elastic adhesive strapping. The application of the elastic adhesive plaster to a wound seems very simple. It is nevertheless necessary to adhere strictly to certain details if the best results are to be obtained: (1) shave any hair bearing skin in the region to be covered, (2) if preliminary treatment (second step) has not been carried out cleanse the skin with alcohol and both the skin and ulcer with ether in order to remove excess secretions, allowing a few moments for drying; (3) elevate the foot by placing the heel on the corner of a chair seat in order to empty the veins; (4) stick two 9 inch strips of elastoplast 3 inches wide lengthwise along the lateral portions of the leg from the ankle to the calf. These need be used only when much edema is present or when an ulcer is so large that it makes a considerable portion of the bandage moist, thus favoring slipping and cutting into the skin. The strips serve to prevent creeping of the circular turns. Another method useful for preventing creasing of the skin is to overlap the spiral turns two-thirds instead of one-third or one-half of their width. They are applied slightly stretched. (5) The foot, ankle and leg are then covered in the order named with spiral turns of the elastic plaster. This is started to one side of the foot and brought across the dorsum to form a figure of 8 over the ankle (see Fig. 3) advanced spirally upward on the leg overlapping one-third to one-half its width to a turn until the calf is reached. Here it is convenient to advance the bandage sharply upward in an

oblique direction in order to complete the highest turn which should be a half inch below the knee cap. It is then run downward again spirally so as to cover the remaining space with a few turns. In applying the plaster the entire ulcer if small enough should be included in one of the turns. This will avoid the cutting of ridges in the granulations by the edges of the plaster. The tension or pull exerted on each turn of the bandage as it is applied should be at least sufficient to stretch the material to the limit of its elasticity, preferably a little greater. With the material we use this will actually register six to eight pounds of linear pull on a spring scale. This expressed in pounds per square inch would be a much smaller figure.

In order to calculate the number of pounds of pressure exerted per square inch of skin surface, we employ a formula which was kindly furnished by Dean Fred J. Lewis of the School of Engineering of Vanderbilt University. Disregarding friction loss which would be overcome by the skin slipping freely over the subcutaneous tissues during application of the bandage, the formula is as follows:

Total Load = Unit pressure × width × diameter or

$$R = \frac{2P}{W.d}$$

P = Recorded load in pounds
R = pressure in pounds per square inch exerted by the bandage
W = width of bandage in inches
d = diameter of cylinder wrapped

Thus if a bandage having a width of 3 inches were applied with a 6 pound linear pull (without overlapping) to a portion of the leg having a diameter of 3 inches, we may obtain the pressure of the plaster on the surface by substituting in the formula as follows:

$$R = \frac{12}{3 \times 3} = \frac{12}{9} \text{ or } 1\tfrac{1}{3} \text{ pounds per square inch.}$$

If each turn of the spiral bandage should be lapped over the last turn by one-half of its width this pressure would be doubled.

It is difficult to impress upon surgeons the necessity of applying the bandage tightly. They are usually afraid of "cutting off the circulation," yet the stagnant venous circulation is exactly what one must "cut off" in order to benefit the tissues. We have never heard of any harm resulting from a too tight application of a bandage unless it be that temporary crease marks occurred from an uneven application of the turns. Rather than being retarded healing is hastened by sufficient pressure. When ulcers fail to improve it is nearly always due to the bandage being applied so loosely that it fails to contract to the reduced size of the leg as the edema subsides. For this reason we agree with Wright in saying that in treating patients with marked swelling of an extremity the initial elastic bandage should be replaced by a tighter one in about 4 or 5 days in order to take care of the great reduction of swelling which in such cases almost invariably occurs. If the extremity is not swollen at the time of the first dressing the initial bandage may be left on for the usual time.

Pain may occur for 4 or 5 hours after bandaging. This is usually not due to too much pressure but rather to the circulation readjusting its paths of flow. This pain is usually temporary and may be relieved by the usual sedatives. It is a good plan to provide a sedative the first night to insure rest for the patient, since he is not accustomed to the bandage and as Wright naïvely remarks "misses the ulcer." It is also necessary to tell the patient that he may go about his duties in the usual way and use his leg, for best results are obtained when the leg is used right along. In case any wound exudate works out through the meshes of the bandage, which is semi-porous, the patient may wash this off with neutral soap and water on a little gauze or a brush. Water will not damage the cloth in any way. After washing, the surface should be dried.

After the first change, it is usually sufficient to renew the bandage only once each 2 to 3 weeks, in fact best results are obtained by infrequent changes. At each re-dressing the elastic adhesive is cut along the side of the leg and foot opposite the ulcer and removed. Any pus underneath is gently sponged away with dry gauze but no other cleansing is done. Before re-application of the bandage the exact size of the ulcer is determined. This is done by tracing the exact size of the ulcer with ink on sterile transparent tissue and by transferring this to the history sheet as a permanent record (see

Fig. 4). If desired the exact area may easily be computed by a transfer to bond paper of known weight per square centimeter (1) and by weighing the exact pattern which is then cut out by scissors. By one of these procedures all guess work is eliminated, for one may easily see with "his own eyes" what progress the wound is making either in healing or in breaking down.

It was through such simple quantitative studies that we have been led to adopt elastic adhesive strapping as the method of choice in the conservative treatment of superficial ulcerative conditions, especially of indolent ulcers of the legs.

The applications of the elastic bandage at stated intervals are kept up for one visit after the ulcer is seen to be completely healed. Following this the patient is told to bathe the leg including the ulcer site with a neutral soap and water, to gently massage it with alcohol, to allow it to dry, and to powder it with borated talcum or zinc stearate. He is also given an elastic cloth bandage of the "Ace" type to wrap around it for slight pressure. He is told to return immediately if any break in the skin should ever occur.

Use of a pressure pad over small deep ulcers. In a few instances in which elastic adhesive was used on large ulcers healing has progressed until a small but deep crater-like ulcer remained. After this stage the healing was very slow, even though the ulcer measured only 5 to 6 millimeters in size. In such cases, we feel that the reason why the epithelium does not grow in to cover the granulations promptly is evident. If the plaster is applied in the usual manner its under surface will be stretched from one edge to the other and will, therefore, be out of contact with the steep sides and deep base of the ulcer. A "dead space" at first filled with air and later with exudate will thus be formed. In order to obviate this the first layer of elastic adhesive is applied under a small amount of tension. Then a pad of gauze or sponge rubber of a size and shape suitable to fit down into the crater-like depression is cut out with scissors. This is applied over the first layer of elastic plaster and is held firmly down into the depression by a second strip of elastic adhesive or a second turn of the bandage under considerable tension. Figure 5 shows the application of such a pad to the ankle. Healing which has been at a standstill has usually proceeded satisfactorily after such a pad is applied. This fact would seem further to show the advantage of pressure in promoting healing. The results of using the pressure pad in such cases are very striking.

Local use of elastic adhesive straps. In cases in which support of the veins of the leg is not necessary and in which only a small superficial ulcer is present a strip of elastic adhesive of a size suitable to cover the ulcer by a fair margin is cut with sterile scissors from the roll, stretched and firmly applied to the skin (Fig. 6). It should not completely surround the extremity. It is usually covered with a few layers of sterile gauze, held in contact by a gauze bandage. Healing under this in selected cases is very satisfactory.

CONTRA-INDICATIONS

So far we have seen personally no cases except acute spreading infections and very deep ulcers in which elastic adhesive strapping seemed contra-indicated. Wright has shown in the case of legs which have been damaged by long-standing venous congestion that "if gangrene supervenes as a result of senile or diabetic arteritis it is not the toes which are affected first but the skin of the leg in the usual ulcer sites." In these cases he feels that compression would be disastrous. Occasionally, a compound fracture of the bones of the leg will result in a projecting knuckle of bone covered only with periosteum and skin. Over such an unprotected prominence, the pressure must be at a minimum in order to prevent ulceration. At first varicose or stocking eczema was thought to be a contra-indication to compression but most of these cases while troublesome at best are less so with compression than with any other line of treatment. In case an eczema seems aggravated by the zinc oxide, one may change over for a time to a bandage impregnated with 5 per cent icthyol or 5 per cent aluminum acetate as recommended by Wright. The important point to recognize is that elastic compression per se does not aggravate the average case of varicose eczema.

ECONOMY OF THE DRESSING

The cost of any technique for the treatment of wounds should be considered as secondary to its therapeutic value. We find, however, that elastic adhesive is very economical to use.

TABLE I.—SHORTENING OF HEALING TIME ACCOMPLISHED BY ELASTIC ADHESIVE

	Time in years ulcer remained unhealed while under treatment by various remedies	Area in square centimeters at time elastic adhesive was started	Healing time in days from application of elastic adhesive	Cost of elastic adhesive treatment until ulcers healed
Ulcer 1	12	15.0	65	
Ulcer 2	10	41.5	51	
Ulcer 3	7	17.5	38	
Totals	29	74.0	154	$13.56
Averages	9.6 years	24.7 cms.	51.3 days	$ 4.52

Table I illustrates its economy from the standpoint of time saved. These three ulcers taken at random from a series of many similar ones had been treated with many different dressings in various dispensaries including our own and failed to heal in an average time of 9.6 years. When subjected to elastic adhesive they healed in an average time of 51.3 days. The saving is obvious. The healing of the ulcer shown in Figure 1 is typical.

Ordinary dressings, by which we mean gauze, ointment, or antiseptic ones require the patient to return at least two and usually three times per week. Elastic adhesive requires only one application each 2 to 3 weeks. This means a great saving to the patient in work hours as well as to the staff physician, nurses, and other dispensary attendants. During treatment the patient is ambulatory, comfortable and able to work. The ulcer loses its foul odor in a few days and the patient is, therefore, no longer a problem to those with whom he associates at home or at work. Oftentimes a patient can retain a job if he has to lose only a morning each 2 weeks which he would be forced to lose if he had to come twice a week for dressings. So much for economy of work hours with elastic adhesive.

A computation of the money saved by this dressing shows rather striking figures. Some of these we briefly quote at the risk of appearing monetary in a medical paper. First, then, it is to be noted that the elastic adhesive technique requires no other article after the preliminary cleansing at the first visit than the bandage itself and a small dry gauze sponge or two at each dressing. Therefore the cleansing and antiseptic solutions, ointments, gauze, bandages and adhesive required for ordinary dressings are almost all saved through its use.

The chief economy in money is made possible through the relative infrequency of dressings required. To get at actual figures one visit in 2 weeks requires only one elastic adhesive bandage, one registration, and one transportation cost against four to six each of the latter two for a similar period with the usual gauze dressings.

At the average price we have paid for the elastic bandage the cost of treatment would be cut to one half by its use ($0.55 compared to $1.17 per week).

To be more specific the three old ulcers described in Table I would have required a total cost for dispensary treatment by the older methods in excess of seventeen hundred dollars in the 9.6 years during which they were treated without cure. With elastic adhesive, they were healed in an average time of 51.3 days, at an average cost of $4.02. It is usually not necessary to keep this new dressing up for more than a short time in order to accomplish healing. However, even granting that it were, the annual saving over the older dressings would be about $32 per ulcer. The average healing time of large ulcers we have shown to be about 51 days. At Vanderbilt, a hospital with 205 beds, the incidence of ulcer of the leg in the Out-Patient Department was 83 per year in 1933. Granting that this incidence was proportionately the same in this country at large in the various approved hospitals[1] having surgical clinics, and assuming that the ulcers would heal only as well by the older methods as usual the annual saving effected for these patients by elastic adhesive in the United States would be well in excess of three million dollars. This money would be saved for precisely the patients who are in the

[1] Hospitals approved for interneships by the Council of Medical Education and Hospitals of the American Medical Association. J. Am. M. Ass., 1933, 101:699.

greatest need of funds. Furthermore a far greater saving would be accomplished for them by the removal of disability and loss of work hours directly attributable to this form of therapy.

PROPERTIES

The chief purpose of using a semipermeable elastic adhesive dressing is to obtain rapid firm healing of ulcerated surfaces. The following properties of the plaster contribute toward this purpose:

a. It is *perfectly adhesive*, which provides the following advantages: Its firm adherence to the skin up to the granulations splints the wound, putting the tissues at rest and preventing the rubbing off or injury of the delicate growing epithelium at the periphery of the wound—an occurrence so usual with other dressings. Its adherence seals the skin away from the ulcer base so that secretions from the wound will not moisten the unsterile skin and then run back to cause reinfection of the granulations. When the dressing is removed desquamated epithelium comes off with it leaving a clean skin and wound surface ready for the next application.

b. It is *semipermeable*. When the bandage is stretched as it should be during its application the small spaces between the threads of the cloth are opened up. This permits the escape of excess secretions through it under slight pressure. The semi-closed system so formed thus retains enough secretion at all times for healing under moist conditions to take place. This may be spoken of as *controlled drainage*.

c. It is *persistently elastic*. This property of elasticity which the plaster exerts continuously 24 hours a day accomplishes several important ends. It provides an excellent firm support for veins which collapses varicosities, thus preventing back pressure and stagnation and improving the nutrition of the part. It provides an elastic support for the tissues which stretches if the leg swells but which again contracts if it shrinks. Thus edema is gradually overcome and by contraction of the swollen leg the size of the wound is diminished. It is interesting in this connection to mention that Carrel has shown that contraction is an important factor in the healing of wounds even in tissues which are not swollen. This point will be discussed in a later report. It further provides a leveling or flattening influence both for the growing edge of the ulcer and for exuberant granulations. Lastly, it provides a smooth immobile contact surface of gummy consistency allowing epithelial cells to grow evenly over the granulations.

d. It is *bactericidal* in effect. The number of organisms is usually rapidly reduced until sterility is reached. This is probably not due to any antiseptic quality of the plaster but to the conditions which it produces locally, as follows: It seals the wound and prevents reinfection. It supports and compresses the veins thus producing a more active circulation to resist germ growth. It favors rapid epithelial growth and thus quickly reduces the size of the field in which bacteria may multiply.

e. It is *hygienic*. Since it is waterproof the patient may cleanse its outer surface with soap and water as often as it becomes soiled.

In using this bandage we must recall that in reality we are employing a material which is a "happy combination" of many past aids to ulcer healing. The virtues of the elastic stocking, of the adhesive strapping advocated by Morison and Beck, of Unna's paste, of the paraffin method of treating burns, and of the use of gutta-percha by Halsted—all are embodied in this one dressing, and yet it seems to have none of their shortcomings or disadvantages.

The properties of the elastic plaster which we have enumerated seem to combine in quite an unusual manner to promote the comfort of the patient and the rapid healing of his wound.

Certain it is that healing results with remarkable rapidity even in ulcers years old which have been indolent and refractory to other forms of therapy. This is such an everyday occurrence that the evidence is indisputable.

RESULTS OF CONSERVATIVE TREATMENT

Elastic adhesive was first employed at Vanderbilt Hospital in the summer of 1931 and has been used constantly ever since. In the 2 years from July, 1932, to July, 1934, 113 cases were treated with this plaster of which

Fig. 1. Progressive healing in varicose ulcer treated by strapping with elastic adhesive.

90 were leg ulcers (i.e., ulcers on the legs, ankles, or feet) and 23 ulcers on various other parts of the body. Case abstracts are at hand in all, but these would consume too much space if given here. Since almost all of the patients have been very co-operative in returning for observation, we believe that the results given are reliable. In only 1 case was a follow-up impossible. In this the ulcer had greatly decreased in size up to the time the patient failed to return.

In 3 cases of leg ulcer, or 3.3 per cent, the lesions improved but failed to heal in 12 months. In 4 more, or 4.4 per cent, the ulcers healed readily but recurrence occurred in a variable period either spontaneously (2 cases) or from some minor injury (2 cases). These recurrences were not serious and all healed promptly when the plaster was applied for a second short interval of time. All other patients of the 113 may be accounted for as cured.

In lieu of a large number of abstracts of case records, we present herewith a few typical ones for the purpose of illustrating points of judgment which we regard important from the standpoint of treatment.

ABSTRACTS OF CASES ILLUSTRATING INDICATIONS FOR CONSERVATIVE TREATMENT AND RADICAL TREATMENT

Case 1. M. W. Colored male of 67 years had a mechanical injury to his lower leg in 1925. This produced an ulcer and other ulcers have appeared and persisted. On physical examination the blood pressure was found to be 180/100. There was also a generalized arteriosclerosis and weak pulsations in the dorsalis pedis arteries. When first seen on May 24, 1933, there were in all six ulcers on the anterior, lateral, and medial surfaces of the leg varying in size from 5 square centimeters to 0.75 square centimeters. Elastic adhesive was firmly but not tightly applied on May 24, 1933, and on June 10, 1933, all ulcers were found to be healed. The leg has been seen after a year and is still firmly healed.

This case is quoted to illustrate the point that elastic adhesive is indicated where ulcers are small; also that a bandage with moderate pressure may be used even though arteriosclerosis should be present and make radical treatment of the ulcers too great a risk.

Case 2. W. W. Colored laborer aged 62. Varicose veins of left leg of 20 years' duration. Contusion of skin of left ankle 15 years before admission. Ulcer of medial surface of left ankle of 15 years' duration. Elastic adhesive was applied January 7, 1933. Five small deep or "pinch" grafts were applied on February 9, 1933. Elastic adhesive was used to complete healing April 7, 1933. The ulcer has remained healed nicely with an elastic cloth bandage to the present time. Figure 7 presents a similar case.

A minor surgical procedure like small deep (pinch) grafting may be used in conjunction with elastic adhesive in order to re-inforce the center of the healing area and so to make it stronger in resisting re-ulceration. This is well demonstrated by this case.

Case 3. G. M. Negro truck driver of 64 years. Varicose veins had been present on both legs as long as he could recall. Thrombophlebitis of veins of right leg occurred in 1908. On admission to Dispensary March 26, 1928, the heart showed hypertrophy. At this time there was elicited a history of "brawny" swelling present in both legs below the knees of several years' duration, giving one the impression of non-filarial elephantiasis. An ulcer almost competely

Fig. 2. Superficial granulating wound from burn of forearm almost entirely healed 8 days after strapping with elastic adhesive. Stain above indicates size of wound at time of first application.

Fig. 3. Method of applying elastic dressing. Heel elevated on corner of chair. First turn over dorsum of foot. Second turn forming figure eight around ankle.

encircled the right leg in its lower third and measured about 15 by 12 centimeters. This had been present for 3 years. The veins were not injected because of the history of thrombophlebitis. The ulcer on the right leg was treated regularly three times a week for 3 years and 11 months with various wet and dry dressings. At the end of this time it was very little smaller. Elastic adhesive was applied to the leg on February 22, 1932. Healing was complete on June 2, 1932. On September 3, one area broke down and formed an ulcer one-half centimeter in diameter but this quickly healed under elastic adhesive. Since then with an ordinary cloth pressure bandage there have been no recurrences.

Thus an ulcer of 6 years' duration, treated intensively at the hospital for 4 years without avail, healed promptly without operation when elastic adhesive was applied. The cost of treatment to the patient for the 4 year period during which he was treated by wet and dry dressings is figured at a minimum of $248.20. This does not include loss of time from work but merely the minimum dispensary registration fees and transportation. The total cost of elastic adhesive treatment including registration fees and transportation up to the time of healing was actually $9.12. In the one case treatment cost the patient $248.00 and the ulcer showed no tendency to heal. In the other his leg was healed before he had spent $10.00.

CASE 4. L. W. Negro chauffeur, aged 25 years, was admitted to hospital dispensary on April 15, 1926. He had a primary luetic sore in 1916. Lues was treated several years later at another hospital. He struck the left shin on concrete curbing in 1922. This wound healed promptly but left lump at site. Pus was obtained on lancing this but the drainage wound then enlarged to form an ulcer which has been present ever since. On admission to hospital extensive complete circular scars were present in the lower thirds of both legs with several open

Fig. 4. Illustrating the use of transparent tissue tracing in determining area of wound during the process of healing.

Fig. 5. Application of pad cut to shape of depression on small, deep ulcers. Pad over first turn of plaster is covered with second turn for pressure.

Fig. 6.

Fig. 7.

Fig. 9.

Fig. 6. Use of elastic adhesive strips on small ulcers.
Fig. 7. Ulcer in aged man which showed slow decrease in size under elastic adhesive. A few small deep grafts applied in dispensary served to accomplish healing.

Fig. 9. Ulcer which after decrease to one-third its original size in 2 months under elastic adhesive failed to heal further in 3 months. Radical operative treatment is indicated.

ulcers on the left (Fig. 8). X-ray films showed slight irregular periosteal thickening of the left fibula. Colon showed chronic ulcerative colitis. Blood Wassermann report varied between negative, anticomplementary, and positive on different occasions. Spinal fluid Wassermann was negative for 0.2 cubic centimeter of fluid, doubtful for 0.5 cubic centimeter of fluid. He received intensive anti-luetic treatment and the usual dry, moist and ointment dressings to leg. Meanwhile similar ulcers formed on his right leg. He was admitted to the hospital but was discharged in a few days without improvement. Dressings were continued along with anti-luetic treatment but new ulcers continued to form and the old ones would crust over at times, only to break down again. When seen in the dispensary on May 24, 1933, the patient presented a pitiable picture. His face was drawn from loss of sleep and pain. The latter resulted from multiple large and small ulcers extending over the larger part of the entire lower two-thirds of both legs (Fig. 8). Both shoes were wet with pus which was running down from these ulcers. He had been having moist dressings for several visits. These were changed to elastic adhesive. The pain was controlled by codeine for a few days and then spontaneously ceased. The exudate which formed came out through the minute spaces in the plaster and was daily cleaned off at home with soap and water. Gradually it became thicker and was released through a few small openings made with sterile

Fig. 8. Multiple bilateral luetic ulcers. Left, before treatment; right, healed condition following use of elastic adhesive.

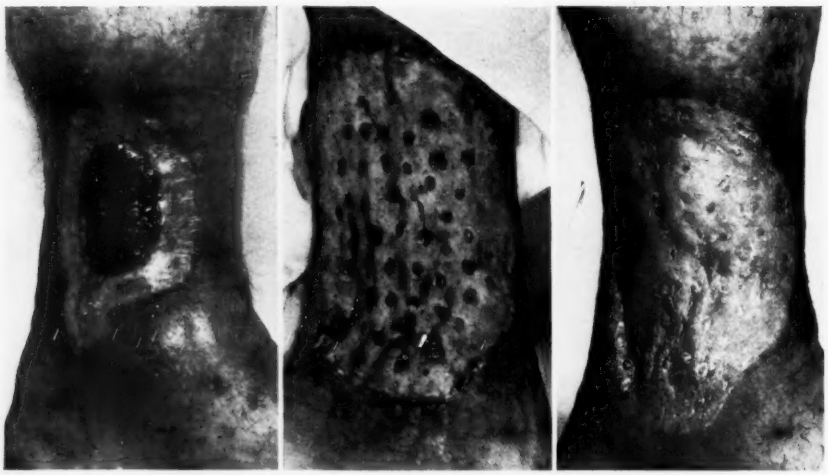

Fig. 10. Treatment by excision and grafting. Ulcer healing slowly 1 month after elastic adhesive was applied. Same area after excision and "sieve" graft. Same area 42 days later.

scissors. The patient's general condition improved as his pain and infection subsided. The ulcers have progressively decreased in size until on September 28, 1933, only pin point spots remained on one leg and pea sized ulcers on the other. All were healed March 17, 1934 (Fig. 8). The itching of the skin was controlled by calamine lotion. There have been no recurrences. The patient was able to work after the first week during which the elastic adhesive was applied.

In this case multiple bilateral syphilitic leg ulcers have healed completely under elastic adhesive dressings after a failure to heal satisfactorily under other remedies during an 8 year period. Anti-luetic treatment was given through most of this period.

CASE 5. C. K. Colored laundress of 48 years. She gave a history of syphilis for which she received treatment "a long time ago." About 4 years before admission, a small blister came spontaneously on anterolateral aspect of the lower third of the right leg. This broke and an ulcer formed which has gradually spread in all directions until it has reached a large size. It has shown no tendency to heal although various types of dressings have been applied. There is no history of varicose veins nor thrombophlebitis. On admission October 17, 1933, a large, fairly deep, foul-smelling ulcer was found almost to encircle the right leg in its lower third (Fig. 9). The skin of the entire lower half of the leg was deeply pigmented except for narrow zones of white scar tissue which surround the ulcer. Both the ulcer base and the tissues around the ulcer were indurated and scarred. The edges were somewhat punched out but not markedly so. The Wassermann was found to be positive. A few moderately dilated veins were seen on the surface of the thigh. The ulcer was thought to be of varicose etiology although syphilis could not be ruled out as a cause. The patient was given anti-luetic treatment. A tracing of the original edge of the ulcer was made and elastic plaster was applied from foot to knee cap. The extreme concavity of the ankle region with respect to the leg and foot made it difficult to keep the bandage from slipping but this was done by using lateral strips placed in the direction of the long axis of the leg. In 2 months the ulcer healed to one-third of its original size. Since then during 3 months time, except for slight variations in size, healing has been at a standstill. The patient has, therefore, been advised to enter the hospital for operation.

We believe that this patient presents an example of the type of ulcer which is so extensive and surrounded by so much scar tissue that healing will be extremely prolonged if indeed it be possible of attainment. The epithelium formed in these cases is very thin and breaks down because of poor nourishment from the scarred bed. Elastic adhesive while it seems to be the best available dressing is palliative rather than curative in such cases. Therefore, excision of the scarred ulcer base and skin grafting should be resorted to.

CASE 6. J. B. A white male laborer of 34 years developed left sided pleurisy in 1929. While still

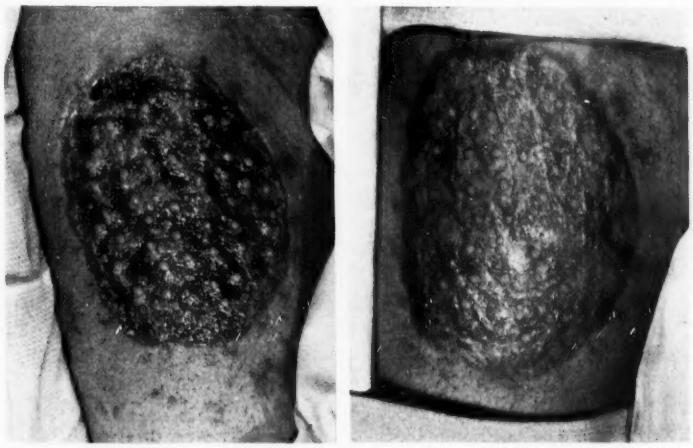

Fig. 11. Treatment by excision and grafting. Left, Site on thigh 14 days after removal of "sieve" graft. Islands are beginning to grow out. Right, Same area 42 days later showing firm healing with pliable skin.

confined to bed, the left leg swelled up and thrombophlebitis was diagnosed. Four months later he scratched his leg and an ulcer formed slightly above his internal malleolus. This enlarged until it was about 5 centimeters in diameter. It healed at times only to break down again. In December, 1932, he states that a "quack" doctor put on some medicine which caused tissue to slough out until the ulcer increased to twice its size. On admission February 9, 1933, a dirty shallow ulcer was seen in the region as described. It was oval in shape measuring approximately 6 by 8 centimeters (Fig. 10). No varicosities were found. After a preliminary treatment for a few days with warm moist boric acid packs, elastic adhesive was applied from toes to knee. At this time the patient was advised to enter the hospital for total excision of the ulcer area followed by grafting. However, he could not make arrangements, and elastic adhesive was continued until April 14, 1933, at which time the ulcer had reached about one-half its original size. On this date the patient entered the hospital. On April 18, 1933, a wide and deep excision of the ulcer bearing area was carried out thus leaving the muscles and some of the tendons bare in the wound. On April 27, 1933, a full thickness "sieve" graft cut to pattern was transplanted from the right upper thigh to the defect. It healed *per primam* and the photograph taken 2 months later shows both areas still stably healed with pliable pigmented skin. The donor site healed promptly with good skin as shown in Figure 11.

We abstract this case to show that while the method of elastic strapping may produce healing of a large ulcer over a joint it may not always be the procedure of choice. Here a full thickness skin graft gave needed re-inforcement in a location frequently exposed to injury. In a young laboring man who had lost time because of several previous recurrences this was of great importance.

INDICATIONS FOR RADICAL TREATMENT

While in the great majority of cases conservative treatment by elastic adhesive has been found to produce unusually rapid healing it has failed to cure a few (3.3 per cent). Also a few of the seemingly cured cases (4.4 per cent) have recurred spontaneously or with minor injury or infection. On the other hand, radical treatment, i.e., by excision and full thickness grafting by the "sieve" method, has resulted in cure in 100 per cent of 15 cases of large ulcer with no major recurrences. This radical method of treatment has been described in detail in two previous articles (2 and 3). It is our belief that radical treatment should be reserved for those patients who cannot be cured by conservative measures in a reasonable length of time. Although no absolute rules should govern individual cases, our experience would seem to indicate that, in the following types of ulcers, conservative measures should yield at once to radical surgery through which alone permanent cure will be reasonably assured:

1. Ulcers due to neoplasm (obvious but included for sake of completeness).
2. Ulcers in connection with elephantiasis.
3. Very large ulcers surrounded and underlaid by avascular scar tissue.
4. Ulcers in which conservative treatment would involve too great a time loss and too great a risk of further disability on account of recurrence (military and industrial group).
5. Ulcers which have failed to heal with conservative measures in 12 months.
6. Ulcers in the region of joints which would heal with scar contractures.
7. Ulcers which after healing have spontaneously recurred or which have repeatedly recurred with minor injury.

This list is not to be regarded as exhaustive.

In the normal course of events the 3 patients whose ulcers we have reported above as failing to heal with elastic adhesive, will be admitted to the hospital for radical treatment which, in our experience, will almost certainly offer permanent healing. If, however, after a fair trial of elastic adhesive such patients refuse radical measures, usually by this time their ulcers are so much improved that the application of the plaster at 3 week intervals will suffice to keep their ulcers small and their legs very comfortable over an unlimited period of time. This no other dressing in our experience will accomplish.

One of these 3 patients who refused to enter the hospital had small deep or "pinch" grafts applied under local analgesia in the dispensary with the result that he is now healed (Fig. 7). This may be regarded as a valuable semiradical procedure.

CONCLUSIONS

1. The cure of large ulcers especially of those on the lower extremities has become extremely important in modern times on account of their increasing incidence.
2. Valuable advances have recently been made in this field along both radical and conservative lines, the radical treatment consisting of complete excision and grafting, the conservative of special dressings.
3. Experience with many large ulcers indicates that all, no matter how extensive, may be cured by treatment directed along one or the other of these lines provided that the underlying cause or causes be discovered and eradicated.
4. In our estimation after a critical experimental and clinical study of its properties and mode of action, elastic adhesive offers an excellent method of conservative treatment, much superior to any other so far developed.
5. Elastic adhesive represents the culmination of an evolution in dressings used in the past for these cases. It combines in one preparation the desirable properties of the Unna's paste boot, elastic stocking, gutta percha tissue, and ordinary adhesive, yet seems to possess none of their disadvantages.
6. Success in the use of this dressing depends upon a preliminary treatment of underlying causes of ulceration followed by an exact technique in its application. If these are carried out it will be found to be extremely comfortable and economical.
7. Prompt healing resulted in 96.7 per cent of 90 chronic leg ulcers treated at Vanderbilt Hospital with elastic adhesive during 1933–1934. During the preceding 20 years a large proportion of such ulcers had shown no improvement with other dressings. All of the cases which failed to heal completely, nevertheless showed definite improvement with this treatment.
8. While elastic adhesive is at present the conservative treatment of choice in ulcers, it failed in this series to cure 3.3 per cent and furthermore some which had healed under it recurred (4.4 per cent). For these ulcers and a few other classes, which are enumerated radical treatment is indicated.
9. Radical surgery by wide excision and full thickness skin grafting by the "sieve" method have proved curative for all of 15 such cases in our hands.
10. If for economic reasons radical measures are not feasible, elastic adhesive will offer the best substitute in treatment.

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A STUDY OF THE RELATIONSHIP OF PREGNANCY TO DISEASE OF THE GALL BLADDER[1]

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THIS investigation[2] is undertaken to standardize and co-ordinate fundamental features in our consideration of a large series of cholecystic disease. Our analysis is concerned with the relationship of the onset of cholecystic symptoms to pregnancy and with the findings elicited by clinical and laboratory tests in the established affliction, as compared to a limited series of partial controls.

Rovsing reported a series of 344 patients with cholecystic disease, more than 80 per cent of whom were mothers. William Mayo reported 3,075 female patients, 90 per cent of whom were mothers, and in whom symptoms referable to the gall bladder became apparent during pregnancy or soon after.

In our service at St. Francis Hospital, 500 patients were operated upon for cholecystitis, 78 per cent of whom had been pregnant.

In the last 500 cases of gall-bladder disease among women at the Elizabeth Steel Magee Hospital exactly 375, or 75 per cent, gave an average history of 4.64 gestations each. There appear 114 miscarriages and 34 abortions in the 375 parous cases, constituting 9 per cent of the total number of pregnancies.

The fact that gall-bladder disease is much more common among women than men indicates that there must be some condition incident to womanhood which is a contributing factor. If 75 per cent of the cases of gall-bladder disease followed pregnancy, then some condition must occur which predisposes to disease of this organ.

Careful study of the history of many of these patients shows that symptoms of gall-bladder disturbance began during pregnancy or soon after, often in the puerperium. The symptoms may be only mild at first. Gas after meals and nausea and vomiting are the outstanding symptoms in the history; they often precede the attacks of severe pain which accompany the presence of gall stones. In many of these patients, careful study of the history will reveal, during the course of pregnancy, evidences of toxemia, such as headache, vertigo, increased blood pressure, etc.

It is difficult to demonstrate that the liver function becomes impaired to such a degree that the character of the bile is changed or that the gall bladder becomes involved. There has been much discussion as to the part the liver plays in the toxemias of pregnancy and also in the so called eclamptic state. Many observers are convinced that, even in a normal pregnancy, the physiological processes of the liver are subjected to extra stress. It is doubtless true that when a pregnancy is complicated by an unusual degree of toxemia, and where all organs are burdened with an extra load incident to the added stress, the liver, from its very nature, is called upon to carry a heavy load. As yet, we know but little about the chemical or physical changes that occur and for that reason a clear understanding of the clinical history and symptomatology is important. A true correlation between our knowledge of the clinical side and what may be gleaned from the chemist or roentgenologist, so far as the functional and physical properties are concerned, may eventually lead to a better understanding of what goes on in the liver and bile passages during pregnancy, and may direct the institution of treatment which might prevent the more serious effects.

It would seem reasonable that under such circumstances the bile may not have its normal characteristics; that the gall bladder and ducts may be exposed to temporary changes secondary to chemical irritation or to infection as a result of lowered resistance. It would appear that in the large percentage of these cases permanent injury persists. Clinical experience teaches that many cases of gall-bladder disturbance are due to poor drainage of

[2] This study of the relationship of pregnancy to disease of the gall bladder was undertaken with the hope that the clinical side of the problem might be correlated with the chemical and X-ray findings and in this way provide more substantial proof of such relationship. It has been made possible through generous aid from the Buhl Foundation of Pittsburgh and the co-operation among the departments of chemistry, gynecology and obstetrics, and radiology.

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the gall bladder and, in a considerable percentage, there is a definite constriction of the cystic duct which interferes with its normal emptying powers. Improper drainage leads to pain and reflex disturbance in the gastrointestinal tract.

The size of the liver indicates its importance in the general nutrition of the body. It has many functions but the principal physiological unit lies in the liver cells, in the opinion of leading physiologists. Much is known about the normal physiology of the liver in relation to the formation and composition of bile, and progress has been made in the study of certain metabolic functions, such as the formation of urea and glycogen. There are other important metabolic processes which are still imperfectly understood, as, for example, its relation to the production of fibrinogen, to the oxidation of fats, and to the detoxication of organic poisons.

One of its most important functions is the disposal of waste products, such as the pigments, cholesterin, bile acids, lecithin, etc. Experiments show that stimulation of the splanchnic nerve diminishes the flow of bile while section of the splanchnic branches may cause increased flow. These actions are explained by their effect on the blood flow through the liver. Since the secretion increases when the blood flow is increased, and vice versa, it is believed that in this case no special secretory nerve fibers exist (Howell).

If the liver is subjected to extra stress, even under normal conditions, it is undoubtedly true that when the patient is handicapped by an excess of poisoning and the inability to withstand the usual strain, injury to the biliary tract may occur. It, therefore, becomes necessary to include the consideration of the possibility of the persistence of such damage after delivery and that careful study and attention be given, not only to the normal pregnancy, so called, but more particularly to pregnancy in which there are signs of unusual toxemia. Infection, stasis, and changes in the cholesterol and calcium content of both bile and blood have all been suggested as etiological factors. Most observers agree that hypercholesterolemia occurs in pregnancy. The work of Aschoff and Bacmeister seems to show that gall-bladder disease occurs in certain instances without signs of infection. Certain individuals are handicapped by poor eliminative power in the liver, and, during pregnancy, when the fetal waste products are added, there undoubtedly ensue changes in both the chemical and physical qualities in the bile which lead to precipitation and predispose to stasis. Investigations by Pribram lead him to conclude that during pregnancy the cholesterin content of the bile was reduced, while that of the blood was increased. In the puerperium he came to the reverse conclusion, that cholesterin content of the bile was higher, while that of the blood was lower. He concludes that increased concentration of bile in the gall bladder does predispose to cholesterin calculi. Hofbauer advances the opinion that hypercholesterolemia in pregnancy is due to insufficient action of the liver cells.

Cholesterol, a non-nitrogenous substance, is a constant constituent of the bile, and it is not supposed to be formed in the liver but it is eliminated by the liver cells from the blood. It is regarded, as far as its appearance in the bile is concerned, as a waste product of cellular disintegration. Lecithin, fats, and nucleo albumin, as found in the bile, represent waste products derived either from the liver or the body at large. The mucilaginous character of the bile is due to the presence of mucin or a nucleo albumin and aside from the fact that it has to do with the viscosity of the bile, little is known about it. What may happen to this particular constituent of the bile under various circumstances remains unknown (Howell).

Much stress has been placed upon the theory of stasis of the bile secondary to enlargement of the uterus with pressure upon the bile ducts. The experiments of Westphal strongly indicate that there is a direct disturbance to the function of the biliary ducts. It is his contention that the action of the gall bladder and its various ducts is dependent upon Oddi's sphincter which derives its nerve supply from the vagus nerve. In his experience slight stimulation of the vagus causes peristalsis of the gall bladder and the biliary passages, thus inducing drainage of bile. Increased stimulation caused sufficient spasm to prevent drainage with the consequent stasis. He calls the

condition a motor neurosis of the biliary passages in pregnancy and is of the opinion that it is a motility neurosis of the biliary passages caused by the increased tone of the vagus and feels that the hypercholesterolemia of pregnancy is responsible for the disturbances of the hormones of the vegetative nervous system.

Experiments by Bacmeister and Havers indicate that hypercholesterolemia in pregnant women is the result of retention of lipoids and is not due to the direct formation of cholesterin within the organism. A pathological condition of the bile itself, secondary to disturbed cell action of the liver, supplemented by periodic interference with drainage of the entire biliary system, would seem to invite disturbed conditions of the gall bladder. This is true especially if to these predisposing causes are added infections of the various types.

So far as the bile pigment is concerned, the simplest explanation, which is supported by recent work, is that the liver cells excrete the bile pigment that is brought to them by the blood just as the kidney cells remove the urea from the blood. On this view the amount of pigment excreted would vary proportionately with the volume of blood flow.

The relief from many symptoms upon the withdrawal of fats from the diet in certain cases is in line with the idea. It is a well known fact that the reduction of meats and fats to which Germany and the other central countries of Europe were subjected during the late war brought about a marked reduction in the number of cases of eclampsia. Couinaud and Clogne, in a careful summary of the work done in hepatic function during pregnancy, have called attention to the antitoxic function of the liver as an important factor, and conclude that in pregnancy the liver protects the maternal organism against itself by the destruction of poisons which result from protein disassimilation and also against poisons from the outside.

It seems reasonable that the various liver function tests, particularly that of Rosenthal, supplemented by radiological study, may eventually assist in clearing up some of these doubtful points, both with reference to the part the liver plays in the toxemias of pregnancy and the crippled state of the gall bladder. A few cases have been reported in which, by means of cholecystography, certain conclusions have been made but no large series in which simultaneous cholecystography and determination of hepatic function during the various stages of pregnancy have been carried out, has come to our attention. It would seem necessary to make a rather wide study of this important subject which involves a considerable number of cases, before any definite conclusions can be made.

It was for this reason that we have undertaken such a study and now have an opportunity to report on a series of 388 patients. There is little doubt that, outside of the state of pregnancy, the method of Graham and Cole gives uniform and constant findings except in unusual instances in which there may be either errors in technique or an unusual reaction of the gall bladder and ducts to some unexplained reaction of the sympathetic nervous system. The questions as to whether a given gall bladder may be somewhat enlarged or just when its emptying time is exactly normal from a physiological standpoint under various conditions, may still offer opportunity for further refinement on the part of the roentgenologist.

Dellepiane believes that in his experience few deviations from normal occur in the first 3 or 4 months of pregnancy but states that with the fifth month there begin to be manifested definite noteworthy modifications in the form and dimension of the cholecystic shadow, in its less intensity and more rapid disappearance of the opacity. He concludes that while modification of the place, form, and volume of the cholecystic shadow observed in pregnancy can be attributed to elements of a mechanical character, it is more difficult to understand deviations from the normal which do not seem to depend upon these facts. He suggests that the complexity of the problem makes further and more profound elucidation necessary.

With reference to the bromosulphalein test for liver function in pregnancy, there seems to be conclusive evidence that it is usually positive in the graver forms of toxemia, especially in eclampsia. Siegel, King, and D'Aprile all believe that the liver function

test in eclampsia seems to indicate the degree of liver involvement. Siegel believes that it is valuable in pre-eclamptic cases in classifying the degree of toxicity and reaction to treatment and also valuable in differentiating nephritic from pre-eclamptic and eclamptic toxemia.

Here again there would seem to be an opportunity for further study. If in the case of an eclamptic the dye retention is always high, it is reasonable to conclude that there is still doubt as to when a given amount of dye retention may be considered abnormal. Whether it may be relied upon in the study of mild toxemias, even as corroborative evidence, is perhaps yet to be decided.

We have 388 cases in the cholecystogram series representing pregnancy from the second month to term and the puerperium for the first 10 days. There was but one case out of the group of 388 who did not have at some time during her pregnancy symptoms referable to the biliary system—heartburn, gas, and nausea and vomiting being the outstanding features. We also have 21 patients from the toxemic clinic and ward, 1 of whom, with a nephritic toxemia, began convulsive seizures 80 minutes after the injection of the dye.

Dye retention is found to be increased in the toxemic group and in the pregnant group in the time in which the placenta is undergoing its greatest development and later when the fetus is assuming greatest growth and development. Fatty acid content of the blood stream in the toxemic group is distinctly elevated, lying at the extreme high level of normal. The fatty acid content of the blood stream in the pregnant group gradually rises, reaching the highest point at around the seventh month. The cholesterol values are within normal limits throughout the toxemic and pregnant groups with the exception of the cases in 2½ and 9½ months groups, and even here we would be very hesitant about calling these values significant or suggestive of any pathology. In the puerperal groups the sixth and seventh days alone are represented by more than 10 cases. Fatty acid content of the blood stream lies at the high level of normal. Cholesterol lies at the high level of normal on the seventh day but is within normal limits on the sixth puerperal day. There is a slight increase in non-protein nitrogen in each instance. Urea content is normal. Sugar is elevated above most of the figures seen in various periods of the prenatal course. Dye retention is less than that seen in a non-pregnant group of cases.

Of the 388 pregnant and puerperal cases studied by roentgen-ray, 288 had reports of some existing cholecystic abnormality. In the order of frequency, the diagnoses made were: enlargement in 172 cases, poor emptying in 48 cases, distortion in 30 cases, small gall bladder in 17 cases, cholelithiasis in 9 cases, and faint shadow, slow emptying, adhesions, and distention in 20 cases.

In conclusion, the studies in this series of cases are concerned with organs, the physiological and pathological functions of which are open to much elucidation. Our knowledge of the influence which the hepatic and cholecystic functions exert over the level of the various constituents of the circulating blood is in many respects pitifully meager. The attempt to correlate concentrations of the various blood constituents with definite disease processes without a logical respect for physiological variations of health and disease is likely to lead us far from the actual truth. On the other hand, it is of importance to determine the range of physiological response to the demands of increased strain of life as compared to the strain of disease. We, therefore, critically analyze our work from the standpoint of a group of known truths.

The basic and signal contributions of Mann and his co-workers demonstrate the tremendous reserve of the liver. The urea formation in the liver of dogs was not appreciably impaired when 80 per cent of the organ was removed.

The increased demands on the constitution of the female during gestation may so alter normal protein physiology that the liver and gall bladder are subjected to insults which prepare the fields for later hepatic and cholecystic disease. We would emphasize our findings as an expression of functional aberration just as the symptoms of nausea and vomiting, gas after eating and epigastric fullness, heartburn, eructations, and headache (in a so

called physiological gestation) are literally billboards of dysfunction.

The use of intravenous sodium-tetraiodo-bromo-phthalein dye must be taken with reserve as a measure of liver function. We should keep the fact in mind that we are injecting a foreign substance into the blood stream, that this substance was originally used by virtue of the physical property of casting a roentgen-ray shadow, and that it is not free of undesirable side effects and may be irritating perhaps to hepatic tissues. We have seen wide variations in excretory response in patients with known cholecystic and hepatic disease. However, the syndrome of a borderline low blood stream sugar, a blood urea content of 8 or below, a dye retention (with standardized dosage) of 14 per cent or above at the end of one-half hour after injection and 8 per cent or above at the end of one hour, and a blood stream fatty acid content on the high level of normal or above, indicates hepatic impairment: the concomitant finding of roentgen-ray and clinical evidence of cholecystic disease presages a stormy cholecystic postoperative course in such an individual. The wise surgeon will resort to pre-operative supportive measures designed to restore depleted carbohydrate reserves in these individuals. It is our experience that the abministration of intravenous glucose to secure this result is a highly commendable measure.

To sum up, of 388 cases we find 56, or 15 per cent, with evidence of well established gall-bladder pathology. The etiology in these cases must have antedated the present pregnancy and consequently must have been due either to previous pregnancy, or to some other cause. The remaining cases may conceivably have originated in the present pregnancy. Of these, we find 44 per cent with the gall bladder definitely larger than normal. This percentage is more than twice as great as was found in an approximately similar number of patients who were not pregnant. It would seem that this change might possibly have some bearing on the frequent development of gall-bladder pathology after pregnancy, since it was the predominating abnormality which we observed.

The relationship between this phenomenon and disease is not apparent from observations made so far. A possible explanation may be that the enlargement is due to stasis of bile in the gall bladder, and this conceivably might predispose to disease.

Our studies did not reveal a large percentage of cases with delayed emptying of the gall bladder, but we do not believe this would rule out stasis as a cause of the enlargement. There might be many explanations to account for this apparent discrepancy. For instance, the customary high fat meal used to test the emptying ability of the gall bladder would hardly be selected as the average meal of a pregnant woman and might give one an erroneous idea as to the every day state of affairs.

Undoubtedly, much more investigation needs to be done on this subject, but we offer the suggestion that the frequent enlargement of the gall bladder which we have noted may have some bearing on the final solution of this problem.

Dr. G. W. Grier gives the following summary as a result of his review of the cases in this study:

The X-ray findings in this series of cases were compiled from the X-ray reports which were made at the time of the examinations over a period of 2½ years. The reports were rechecked just previous to the writing of this article and very few changes were made, although naturally at this later period the authors have had the benefit of 2 more years of experience in the work of cholecystography.

The reports were made without a knowledge of the clinical aspects of the case and without any pre-conceived ideas of what we should find. As a matter of fact, many of these cases were passed on by the roentgenologist without his knowing that they belonged to this series and no effort was made to keep tab on what the findings were as we went along.

The interpretations, therefore, were not biased in that respect, and we are confident that we did not read into the roentgenograms something we were looking for, since, as a matter of fact, we had no idea what we were looking for.

Summarized, the findings are as follows:

	No.	Per cent
Normal gall bladder	151	38½
Abnormal gall bladder shadow	208	54
No shadow	29	7½

Abnormal	No.	Percentage of total
Enlarged gall bladder	172	44 1/3
Poor emptying gall bladder	48	12 2/5
Stones	9	2 1/5
Small contracted gall bladder	17	4 2/5
Deformity of gall bladder	30	7 2/3
Faint shadow	20	5
No shadow	29	7 1/2

We see at once that less than half the cases had a definitely normal gall bladder. Of those that were abnormal we might deduct those that had stones, deformities that could be attributed to adhesions, or were small and contracted indicating marked disease of the gall-bladder walls, since it seems that these must have been diseased before the beginning of the present pregnancy. I say this because it is probable that more than 9 months would be necessary to produce these lesions. If we accept this, then the present pregnancy could hardly be responsible for them.

However, they may have been produced by previous pregnancies and a consultation of our records shows that of:

9 cases of stone, 9 had previous pregnancy
17 cases of contracted gall bladder, 9 had previous pregnancy
30 cases of deformed gall bladder, 12 had previous pregnancy

A total of 30 of 56 cases, therefore, have had a previous pregnancy which, of course, might have been the etiological factor in their gall-bladder pathology. These 56 cases might be said to show direct and definite signs of pathology.

The remaining signs of enlargement, faint shadow, and poor emptying might be considered debatable, first as to degree since this is a question of judgment; and second, it might be questioned whether they are actually evidence of pathology at all. However, the large percentage of cases showing these findings makes one hesitate to dismiss them as having no significance.

Of the pregnant women of this series 44 per cent had a demonstrable enlargement of the gall bladder which should call for some explanation.

As a check against these findings, I have examined the records of the Pittsburgh Diagnostic Clinic where no pregnant women have been examined. Out of 458 cholecystograms there were 90 that showed enlargement, a percentage of 19⅔ per cent; and there were 47 showing poor emptying, a percentage of 10⅓ per cent.

While the percentage of those showing poor emptying is about the same, it will be noted that the percentage of enlargement is very much greater in the case of pregnant women.

Poor emptying which was recognized in a much smaller percentage (12 per cent), but still a considerable number of cases, might be considered as a natural accompaniment of enlargement, since it must result in the latter condition if it persists for some time.

Faint shadow which has been reported by Levyn as a characteristic finding in gall bladders of pregnant women was present in only 5 per cent of our cases.

The finding of failure of visualization of the gall bladder we have ignored entirely, since there is always the possibility that this finding may be due to errors in technique. Naturally, in this kind of a case, the errors in technique would be greater than normal, and many of these patients were unable to speak English or were of such a low order of intelligence that they could not appreciate directions. So we feel that it would not be fair to accept failure of visualization as having pathological significance.

Roentgen-ray studies of cholecystitis offer definite and helpful diagnostic adjuvants, but the roentgen-ray plate cannot take the place of cortical cells in the estimation of the pathologic status of these cases.

There is a demonstrable similarity between the functional blood stream expressions of aberration encountered in certain phases of pregnancy as compared with various degrees of cholecystic and hepatic pathology. From the higher incidence of gall-bladder disease in women who have borne children, from a comparison of clinical symptomatology and the functional metabolic disturbances seen in cholecystic disease and in pregnancy, we are led to the conclusion that the institution of measures directed toward the control of func-

tional disturbances in pregnancy might contribute to a lowering of gall-bladder disease. This statement is made in the face of the knowledge of the high regenerative power of hepatic tissue.

The great temporary relief experienced by patients with chronic gall-bladder disease by the administration of diets containing a maintenance protein, a low fat, and a high carbohydrate encourages the belief that much permanent injury to hepatic and cholecystic tissues may be obviated at its inception. If the correctness of these concepts may become fact, the sphere of the obstetrician's usefulness in the field of internal medicine will be greatly extended.

Every pregnant woman who shows evidence of toxemia, as manifested by persistent nausea, vomiting, gas after meals, etc., to an unusual degree should suggest to the physician or obstetrician that he should not be unmindful of the effect upon the biliary tract, and in the treatment should appreciate the additional responsibility in the prevention of permanent disease of this organ.

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PEPTIC ULCER

An Experimental Study[1]

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THE etiology and pathogenesis of peptic ulcer has offered one of the most perplexing problems of modern medicine. In an endeavor to elicit the facts in ulcer causation an enormous literature has been built up about the ulcer problem.

REVIEW OF LITERATURE

Reviews of this literature have been brought up to date from one period to another by a number of authors. MacCullum reviewed the earlier experiments which were primarily concerned with the production of mucosal defects. Later McCann (26) made a comprehensive review of the literature which dealt with the production of ulcers by toxic manifestations, by vascular occlusion, on a bacteriological basis and finally on the basis of trophic and glandular disturbances (neurogenic theory). These authors finally concluded, that, with the many types of experimental procedures employed, the occurrence of ulcers was variable. Contradictory results were reported. Of the particular lesions that did develop, the smaller ones healed spontaneously, whereas those that were too large led to the death of the animal. The production of a true, chronic, indurated lesion, was the exception and not the rule.

In 1913, Boldyreff introduced a new thought which was to become monumental in the history of experimental peptic ulcer. By means of animal experimentation, he was able to show that the concentration of hydrochloric acid, as secreted by the stomach was from 0.5 per cent to 0.6 per cent hydrochloric acid. Normally this concentration was reduced from 0.15 per cent to 0.2 per cent by the saliva, ingested food, mucous secretions of the stomach and reflux duodenal contents. Of these he believed the refluxed duodenal contents, and more specifically the pancreatic juice, to be the most important factors.

Following the promulgation of this theory of "the self regulation of the acidity of the contents of the stomach," the problem of ulcer causation for the first time came to be attacked with a definite working hypothesis. Attention was now focused on the neutralizing influence of the digestive juices found in the duodenum. In an endeavor to evaluate the importance of any one or combination of these juices, multiple ingenious surgical procedures have been devised. But however fantastic or simple a procedure employed, the objective has always been the same, namely: to exclude one or more of the duodenal contents from the upper gastro-intestinal tract. It was thought that, when the normal neutralizing mechanism was removed in part or in its entirety, the acid-pepsin fraction of the gastric juice would be permitted to exert its deleterious influence on the intestinal mucous membrane.

Any one of these methods could be classified under one of three groups:

1. Those dealing with pancreatic exclusion.
2. Those dealing with exclusion of the duodenum, with or without its digestive juices.
3. Those having as their only objective—biliary exclusion.

Pancreatic exclusion. The problem of excluding the external secretions of the pancreas has been approached by injecting substances to inhibit the flow of pancreatic juice, or by tying off or cannulating one or both pancreatic ducts as they enter the duodenum. Jona (16) injected "extracts of decomposing animal tissues" and noted the occurrence of gastric and duodenal ulcerations which he attributed to the inhibited secretion of saliva and pancreatic juice. To add additional evidence in favor of this theory he then tied the main or both of the pancreatic ducts in dogs and subsequently demonstrated duodenal and occasionally gastric ulcers (17).

Ivy (14) studied the incidence of ulcers in one thousand necropsies which included healthy dogs, parathyroidectomized dogs, dogs with ligated pancreatic ducts, and suprarenalectomized dogs. Of especial interest were

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the 24 dogs with ligated pancreatic ducts. Four of these revealed superficial erosions. One animal, whose pancreatic ducts had been ligated 5 months previously, showed a typical, deep, eroded, indurated ulcer in the first part of the duodenum.

Gallagher studied experimentally the possibility of intestinal clamps causing subsequent ulcer formation. The pressure applied by the clamps was carefully measured in various surgical procedures. Following the application of sufficient pressure, acute ulcers could be demonstrated at the line of insult. The acute lesions healed quickly with the formation of scar tissue and a moderate dilatation and thinning of the wall of the duodenum at the site of the healed lesion. In one group of animals the main pancreatic ducts were ligated. The traumatic ulcers in these animals showed a delayed healing time. This delay in healing was attributed to the partial removal of the normal alkalinizing mechanism and to the cachexia of the animals.

More recently Elman (8) and his associates have carefully studied the relation of pancreatic exclusion to ulcer causation. These workers first provided a means of collecting the total external secretion of the pancreas under sterile conditions. In the uncomplicated instances the animals died in 5 to 8 days with marked asthenia. At autopsy it was noted that the stomach was small and contracted, particularly in the pyloric region. The hyperemic mucous membrane was thrown into deep folds and showed small erosions or ulcerations. Another group of studies (6) were then made under the same conditions to show the neutralizing influence of pancreatic juice on the gastric acidity. Their conclusion confirmed the original observation of Boldyreff that the reduction of the gastric acidity is in part due to the reflux of the alkaline pancreatic juice into the stomach. With this as a working hypothesis another series (7) of animals were prepared and life maintained as long as possible by careful feeding and the daily administration of 500 cubic centimeters of Ringer's solution intraperitoneally. Spontaneous peptic ulcers were observed in 6 dogs that were kept alive and in good condition for 13 or more days.

Matthews and Dragstedt based their work on the same hypothesis, but employed a new method in constructing permanent pancreatic fistulas. By the daily administration of salt solution their animals were kept alive for longer periods. In these experiments one, or more, chronic ulcers were found in the duodenum near the anastomosis with the stomach.

As a control study for their experimental work on peptic ulcer, Berg and Jobling employed dogs with pancreatic fistulas. Nine animals were observed for periods ranging from 17 to 52 days, respectively. They were unable to demonstrate any lesions in the stomach or duodenum.

From these contradictory reports, it becomes at once obvious that pancreatic exclusion as a method of inducing experimental peptic ulcers is at its best still a problem.

Duodenal exclusion. Other workers interested in the experimentally induced ulcer have employed the second type of surgical procedure; namely, exclusion of the duodenum with or without its digestive juices. The titles of "Surgical Duodenal Drainage" and "The Short-circuiting of the Duodenal Contents" have been coined to identify this type of systematic manipulation.

Bickel, quoted by Exalto, was interested in the study of biliary and pancreatic secretions in dogs with gastro-enterostomies. The procedure consisted of performing a gastro-enterostomy, extirpating the duodenum, ligating the pylorus and then transplanting the common bile duct and pancreatic ducts into the skin. Four and one-half weeks later the animal died following the perforation of a postoperative jejunal ulcer.

Later, Exalto, interested in the occurrence of gastrojejunal ulcers following gastro-enterostomies noted that although these particular lesions had been observed frequently in man, their occurrence remained unexplained. Because no experimental attack had ever been made on this problem he undertook this particular investigation. He performed gastro-enterostomies on a series of 7 dogs. When the dogs recovered from these operations combined acid (50 cubic centimeters 1 per cent hydrochloric acid introduced by a stomach

tube) and meat feedings were instituted. Three came to be autopsied because of secondary complications at 36, 160 and 197 days, respectively. There were no gross ulcerations or microscopic evidence of inflammation. The 4 remaining were living and well at the end of 222 to 289 days and failed to show any evidence of gastro-intestinal ulceration. From this he concluded that gastro-intestinal ulceration could not be produced by doing a gastro-enterostomy and following with acid meat feedings.

Unable to induce postoperative ulceration by these procedures, Exalto then resorted to the Y-form of gastro-enterostomy. Four such animals were given acid meat feedings after operation. The first dog was sacrificed on the twenty-ninth day after operation because of a mistaken diagnosis. An ulcer was found in the blind end of the jejunum near the anastomosis. The second dog, because of anorexia, nausea, and vomiting, was sacrificed on the fifty-eighth day. There was no evidence of inflammation or ulceration. The 2 remaining animals died of perforated jejunal ulcers on the seventy-first and seventy-third days, respectively.

In the ulcer free animal sacrificed on the fifty-eighth day, bile was demonstrable in the stomach and intervening duodenal segment. To this presence of bile, Exalto attributed a neutralizing influence which he believed prevented ulceration of the gastro-intestinal mucosa. He then devised a method whereby the bile could be prevented from entering the prospective areas of ulceration. Three animals were prepared so that the jejunum was anastomosed to the stomach, the pylorus excluded, and then the distal end of the isolated duodenal segment implanted into the cecum. The first two were fed the acid-meat mixture after operation, and died of perforated jejunal ulcers on the twenty-first and thirty-eighth days, respectively. At this point it was noted that the ulcers seem to develop prior to the acid meat feedings. In answer to this the third dog was fed a mixed diet after operation and died of a perforated jejunal ulcer on the thirty-ninth day after operation.

Later Kehrer, with a different motive, studied experimental ulceration. His thought was to induce a prolonged and intermittent closure of the pylorus and note the effect in ulcer causation. In 15 dogs, he ligated the common bile duct, performed a cholecystileostomy, destroyed the duct of Wirsung, and then anastomosed the duct of Santorini into the lower ileum. Nine animals failed to reveal any gross changes in the stomach after 9 to 159 days. In the 6 other dogs, definite microscopic changes of inflammation, erosion or ulceration were demonstrated after 9 to 106 days after operation. These changes were attributed to a hypermotility and increased acidity.

More recently, Mann and Williamson employed the same principles with a modified procedure in that the contents of the duodenum was drained into the terminal ileum. Mann and Williamson, and subsequent workers have repeatedly observed the occurrence of peptic (jejunal) ulcers in a large percentage of animals prepared in this manner. Through the efforts and numerous valuable contributions of Mann and Williamson, this type of surgical manipulation has become almost a standardized method of inducing peptic ulceration in the experimental animal.

Biliary exclusion. In attempting to solve this problem of ulcer causation, there has been a third and significant group of workers, namely, those interested in the relation of biliary exclusion to ulcer causation.

Whipple (32, a) was the first to note the occurrence of peptic ulcers following complete exclusion of bile. Primarily interested in the output of bile pigment he prepared a series of animals by first ligating the common bile duct and then suturing the open fundus of the gall bladder to the skin. In this study he incidentally noted a deep penetrating duodenal ulcer in one of the animals that came to autopsy on the thirty-fourth postoperative day.

Kapsinow, interested in the experimentally induced ulcers following biliary exclusion, criticized the work of Whipple on the assumption that infection might ascend by way of the gall-bladder sinus. To obviate this objection, he first performed a cholecystnephrostomy and then at a second operation ligated and divided the common duct. After this

second operation, the course of the animals was progressively down hill. They lost weight, refused food, and developed tarry stools. When autopsied or sacrificed at the end of 2 or more weeks the frequency of duodenal ulcers was high. Of this series of 43 animals, typical duodenal ulceration was demonstrable in 17 (40 per cent).

The next report on the incidence of experimental peptic ulceration following biliary exclusion was made by Berg and Jobling. These workers effected the complete exclusion of bile after the method of Rous and McMaster. Twenty-three animals were studied and divided into three groups. The first group comprised the uncomplicated biliary fistulas, the second group the biliary fistulas followed by biliary obstruction, and the last group those with biliary obstruction. Of this series, ulcers developed in 60 per cent of the animals. In this study it is interesting to note that the dogs with complete biliary obstruction developed ulcers just as did the dogs with either temporary or permanent drainage of bile.

That peptic ulcers do develop in animals with obstructive exclusion of bile, is substantiated by the reports of other workers. Malkoff, quoted by Bogoras stated that, by ligating the common bile duct, gastric and duodenal ulcers could be subsequently observed. Simnitzky, also quoted by Bogoras, ligated the common bile duct in 5 dogs. In 3, ulcers were observed in the pylorus and duodenum.

Hosomi, working on plastic transplants into excised portions of the common bile duct, incidentally noted that many animals at autopsy presented peptic ulcers. Then to study the pathogenesis of these ulcerations, he repeated his experiments on 15 animals. Homogeneous transplants from the carotid artery were sutured into surgically made defects of the common duct. Seven of these animals (46 per cent) revealed either mucosal erosions, submucosal hemorrhages, or ulcerations in the stomach or duodenum. Careful postmortem studies revealed that in 2 animals there was a definite complete mechanical obstruction. In 3 animals there was a definite constriction of the biliary passage. From these observations he concluded that complete or partial interference of the flow of bile into the duodenum was an important factor in the pathogenesis of these ulcerations.

Bollman and Mann, in studying the experimental control of ascites in a large number of dogs noted that many animals were lost to the original study because of the early development of perforated duodenal ulcers. Their operative work consisted of producing an acute mechanical biliary obstruction. In 64 animals, acute, subacute or chronic gastroduodenal ulcers developed in 5 to 295 days. Twenty-three other animals died in 22 to 195 days but failed to show any gross evidence of ulceration. It might be added, however, that of these 23 animals, the cause of death in 8 was attributed to gastro-intestinal hemorrhages.

That the loss of bile *per se* plays a rôle in the experimental production of peptic ulcers is further suggested by the work of Weiss and Hubster. A series of experiments was carried out in an attempt to discover the cause of gastroduodenal ulcer. In one group of animals, the gall bladder was anastomosed to the ileum 30 to 40 centimeters beyond the cecum. Their thought was that there would not be a disturbance of the calcium metabolism such as would follow the complete external loss of bile. There followed an acute inflammation of the antral and duodenal mucosa interpreted as signifying a pre-ulcerous stage.

Pausing at this point to rationalize on the mentioned contributions to the problem of ulcer causation, certain deductions seem inevitable: (1) that pancreatic exclusion as a means of inducing experimental peptic ulceration is as stated, still a problem; (2) that by short circuiting the duodenal contents to the lower bowel, peptic ulceration will follow in a large majority of instances; (3) that when there is a complete exclusion of bile from the entire, or at least the upper intestinal tract, peptic ulcers occur in a large majority of these animals.

That ulcers may be produced experimentally by either shortcircuiting the duodenal contents or effecting biliary exclusion the question arises: "Why do peptic ulcers develop in these animals." The answer to this

question may lie in the proper analysis of the distorted physiology following these operative procedures. In a consideration of the short-circuiting method, the following changes are to be considered.

1. That appreciable trauma is inflicted on the gastro-intestinal mucosa is unquestionable. This is especially true where mechanical clamps are used. Attempts to evaluate the importance of this factor have been made by Gallagher, Exalto, and others. The conclusions derived in each instance are not only confirmatory of one another, but indicate that acute traumatic lesions inflicted on the normal gastro-intestinal mucosa heal rapidly; usually within 10 days.

2. By dividing the stomach at or just proximal to the pyloric sphincter, the normal sphincter mechanism is either interfered with or completely destroyed. That the normal function of the pylorus with its sphincter is disturbed in the ulcer patient is well appreciated by many clinicians. Some clinicians (11) feel so strongly about this particular disturbance as to say "When we can effect a 'dumping stomach' in the ulcer patient we shall have found, or can find, the cure for ulcer disease." This same point has been equally well appreciated by many surgeons. Innumerable gastro-enterostomies have been performed on ulcer patients in hope of providing a means for the rapid emptying of the stomach. The questions now arise: How is this disturbance of the normal pyloric mechanism brought about in the ulcer patient? What relation does the extirpation or impaired function of the pyloric mechanism bear to the production of the experimental ulcer? At present these questions remain unanswered not only because of our inadequate knowledge of the normal pyloric mechanism, but also because of the etiological factors instrumental in producing peptic ulcer disease.

In the animals subjected to this shortcircuiting operation not only is the function of the sphincter mechanism impaired, but by joining the jejunum to the stomach by an end-to-end suture the lumen at the site of the anastomosis is appreciably narrowed. Further, this narrowed lumen lies just distal to the powerful musculature of the pyloric antrum. As a consequence of this distorted arrangement at the gastro-intestinal junction, the acid gastric chyme is forcibly ejected by the powerful antrum through the narrow outlet of the stomach into a more vulnerable region of the small intestines. Mann appreciated this fact and stated that he believed this to be one of the factors operative in producing ulcers in these animals. More recently however, it has been demonstrated (15, 24) that when the muscular pylorus was resected and a wide anastomosis made between the stomach and jejunum, the incidence of ulcers was equally high. This suggests that even when the "motor drive" effect is dissipated, ulcers continue to occur due to other factors.

Another end-result of the shortcircuiting operation is that the jejunal mucosa is brought into direct contact with the gastric juice. In this connection Ivy and Fauley (15) have demonstrated that the jejunal mucosa is more susceptible than the duodenal mucosa to the deleterious influence of the gastric juice. That this fact alone is insufficient to account for the occurrence of ulcers in these animals can readily be appreciated by again referring to the work of Exalto. Exalto, in his first approach to this problem, performed gastro-jejunostomies on normal dogs and then gave acid-meat feedings. He never was able to demonstrate the occurrence of either gastro-jejunal or jejunal ulcerations.

As mentioned, the tenor of the work on duodenal exclusion has been that the acid of the gastric juice, not being neutralized by regurgitated duodenal contents, gives rise to ulcerations in the unprotected mucosa. That the acid-pepsin fraction of the gastric juice is of great importance in the production of peptic ulcers must be conceded because of the tremendous amount of clinical and experimental evidence adduced in its favor. That it is not of paramount importance is being appreciated more each day by modern clinicians. It is more important to note that its actual occurrence in the experimental animal is becoming questionable in the light of recent investigations. That the variations in gastric acidity do not depend on the regurgitation of alkaline duodenal contents is suggested by the works of Berg and Jobling, MacLean and

Griffiths, McCann (25), Yesko, Ivy (15), and others.

Finally the last change to be considered is the actual loss of the digestive juices of the duodenum to the upper intestinal tract. This loss comprises that of three digestive juices; the secretions of the duodenal mucosa, the bile and the pancreatic juice. Because of the technical difficulties encountered in attempting to isolate or exclude the secretions of the duodenal mucous membrane, we are unable at the present time to account for the rôle played by them in digestion or the significance of their loss in the experimental animal. That the loss of pancreatic juice questionably leads to experimental ulceration has been referred to above. In the ultimate analysis then of this shortcircuiting procedure it appears that the factor of paramount importance is the loss of bile to the upper intestinal tract.

That the loss of bile is so instrumental in inducing experimental ulcerations is readily appreciated from the work done on biliary exclusion. The particular method used in excluding the bile appears to be of minor importance. Whether the flow is obstructed, excluded to the exterior, drained into other viscera, or lost to the lower intestinal tract, the results are the same.

STATEMENT OF THE PROBLEM

The purpose of this study was to show that the loss of bile was of paramount importance in inducing experimental ulceration. The plan was to collect externally the bile as secreted by the liver and then to feed back to animal the bile that had been collected. The animals were then studied for the development of ulcers. To make the study comprehensive and in addition to try to determine whether the changes induced experimentally are comparable to the changes observed in man, all phases of this work were supplemented by careful histological studies.

MATERIALS AND METHODS

Three groups of animals were studied. The first group consisted of 2 normal dogs sacrificed to act as control studies. The second group consisted of 5 animals with complete external biliary fistulas. The fistulas were prepared after a modified Rous-McMaster procedure under drop ether anesthesia and with aseptic surgical technique. The bile as secreted by the liver was collected externally in a sterile condom but was not fed back to the animals. In the third group of 3 dogs, the collected bile was fed back to the animals after being mixed with the laboratory stock diet.

In every animal when the condition became poor it was sacrificed to insure fresh material for microscopic study. Pieces of tissue were taken from all portions of the stomach, duodenum, jejunum, and liver. The tissues from the stomach, duodenum, and jejunum were fixed in alcohol and prepared for staining with muchematein, hematoxylin, and eosin and van Gieson's stain. The blocks of liver tissue were fixed according to routine methods in Zenker's solution and formalin and prepared for staining with hematoxylin and eosin, van Gieson's stain, and a modified Herxheimer's stain for fat.

RESULTS OF EXPERIMENTS AND OBSERVATIONS

In the following pages are abstracted protocols of the animals used in this work. Following each record the observations made from histological studies are given.

Normal (Control) Animals

Dog Normal No. 1. Medium sized, healthy, male mongrel. After a large feeding of chopped meat, food was withheld for exactly 24 hours and then the animal was sacrificed. At autopsy the abdominal viscera were grossly clean and normal. Microscopic studies revealed a normal distribution of glandular structures. In the muchematein stained section all of the mucous secreting cells were packed with deep blue staining granules. The nuclei appeared to be flattened against the basal border of the cells. On the surface of the entire gastric mucosa there was a film of mucin.

Dog Normal No. 2. Small, active, male mongrel. This animal was given a large feeding of meat early in the morning and again early in the afternoon. The purpose was to study the cells after they were exhausted by physiological digestion. In comparison with the previous animal the chief changes were noted in the muchematein preparations. In these sections the mucous secreting cells presented the aggregation of deep blue granules only in the distal theca of the cells. The nuclei were large, ovoid, and occupied a more central position. The intermediate zone presented only a few lightly stained granules.

The surface of the mucosa was covered by a thin film of mucus.

Biliary Fistula Dogs Without Bile Feedings

Dog No. 4-4. Medium sized, brown and white mongrel weighing 16.8 kilograms. Complete external biliary fistula was made. Recovery after operation was uneventful and animal remained in good condition for 4 days with fair appetite and free drainage of bile. Thereafter condition was progressively down hill and drainage of bile became less to complete cessation by the eighth day. It was sacrificed on eighth day.

Autopsy. Weight 13.6 kilograms. Diffuse seropurulent exudate in peritoneal cavity. The only suggestive pathological areas were limited to the duodenum. Mucosa was reddened in large placque-like areas suggesting an incipient duodenitis.

Microscopic examinations. The histological findings were practically identical to the findings in dog No. 5-4 so that a single description will suffice for both animals.

Dog No. 5-4. Medium sized, tan female mongrel weighing 21.3 kilograms. Complete external biliary fistula was made. Immediate postoperative course was uneventful. Drainage of bile was free but animal refused all food after the first day. Condition was rapidly down hill and it was sacrificed on the eighth day.

Autopsy. Dog weighed 16.4 kilograms. The peritoneal cavity was grossly clean. The pathological area was limited to the pylorus along the greater curvature. Here there were small areas of a reddish brown color suggesting petechial mucosal hemorrhages.

Microscopic examinations. No changes were noted in the cardiac and fundic gland regions. In the pyloric glands beginning inflammatory and degenerative changes were noted. These changes were limited to the superficial layers of the mucosa and occupied discrete areas of about 1 to 2 millimeters in diameter. The margins of these altered zones gradually blended into the normal glandular mucosa. The characteristics of these pathological areas were the presence of an inflammatory exudate, loss of the surface epithelium, destruction of the gland neck epithelium, and occasional cystic dilatation of the gland necks. These changes were best seen in the hematoxylin and eosin stained sections as illustrated in Figure 1. In comparable sections stained with muchematein it was interesting to note that regardless of how much of the cell cytoplasm had been destroyed the remaining portion took on the characteristic deep blue staining reaction.

Paralleling these alterations in the pylorus, similar changes were noted in the mucosa of the duodenum. In the duodenum, however, the presence of a surface inflammatory exudate was not as constant as in the pylorus. The surface destruction also differed in that the changes varied from a mere loss of covering epithelium to complete erosion and loss of the villi. The changes in the jejunum were identical but present to a less marked degree. These changes were also best demonstrated with hematoxylin and eosin staining as illustrated in Figure 2.

The only discernible change in the liver was a fatty degeneration of the hepatic cells lying about the central vein of each lobule.

Dog No. 6-4. Large tan, female mongrel weighing 23.1 kilograms. Complete external biliary fistula was made. For 3 days, the condition of the animal remained good, ate with a fair appetite, and there was a free drainage of bile. Beginning with the fourth postoperative day, her appetite became poor, drainage of bile scanty, and the course progressively down hill. It was sacrificed on the thirteenth day because of the poor condition.

Autopsy. The peritoneal cavity was clean and the drainage tube was well walled off by omentum. While examining the liver a right sided subdiaphragmatic abscess was broken into. The liver in this region was deeply injected and edematous. In the mucous membrane of the pylorus, there were small punctate areas of brownish discoloration again suggesting petechial hemorrhages. The mucosa of the duodenum and jejunum were diffusely reddened. On the posterior wall in the first inch of the duodenum were four small (one-half centimeter) superficial erosions.

Microscopic examinations. No changes were noted in the cardiac, fundic and pyloric gland regions. Essential findings were in the duodenum. In the superficially eroded areas described above there was almost a complete destruction of the mucosa. The intestinal villi were entirely destroyed. The simple tubular gland portion revealed marked interstitial round cell infiltration with varying degrees of involvement. In some sections only the most superficial portions of the glands were eroded whereas in other sections (Fig. 3) almost the entire glandular layer was destroyed. In the Brunner gland regions there was a dilatation of secreting acini and collecting tubules. This dilatation ranged from almost normal appearing acini to almost unbelievable proportions as illustrated in Figure 3. In the muchematein preparations any remaining portion of cell cytoplasm (of mucus secreting cells) took on the characteristic deep blue staining reaction. The remaining non-eroded portions of the duodenum revealed microscopic evidence of a low grade duodenitis as described and illustrated under Dog No. 5-4. In the jejunum similar but less marked changes were demonstrable.

Fatty changes in the liver were similar to the changes described for Dog No. 5-4.

Dog No. 9-4. Small, tan, long haired, female mongrel weighing 11.3 kilograms. A complete external biliary fistula was made and drainage of the bile was free and the animal ate well for 3 days. Thereafter appetite steadily diminished. Course was progressively down hill and dog was sacrificed on fifteenth day after the drainage tube was pulled out.

Autopsy. Dog weighed 7.7 kilograms. The peritoneal cavity was clean except for several small

periductal abscesses near the hilus of the liver. Multiple pin-head-sized brownish spots were found in the pyloric mucosa. In the first part of the duodenum there were three small ulcers about one-half centimeter in diameter. The ulcers were shallow with flat edges. The remainder of the duodenal and jejunal mucosa showed a diffuse reddening.

Microscopic examinations. No changes were noted in the cardiac, fundic or pyloric regions. The chief findings were in the ulcerated areas of the duodenum. The lesions appeared active with sloping edges formed by the mucosa proper. The bases of the lesions were made up of an inflamed débris resting on the submucosa. The submucosa was infiltrated with round cells, appeared edematous, and seemed to bulge up into the base of the ulcer as illustrated in Figure 4. The remaining portions of the duodenum and jejunum revealed usual changes of duodenitis and jejunitis as referred to. The liver showed fatty changes as previously described.

Dog No. 2-4. A large, short-haired, tan female mongrel weighing 20.9 kilograms. Complete external biliary fistula was made. As in the other dogs this animal ate well at first. After the first few days the animal lost her appetite and her condition became poor. She was sacrificed on twenty-third day after operation.

Autopsy. Dog weighed 13.6 kilograms. The peritoneal cavity was clean. Petechial areas of discoloration were noted in the pyloric antrum. In the duodenum and jejunum there was a diffuse reddening of the mucosa. Other than for a somewhat mottled appearance the liver appeared normal.

Microscopic examinations. No changes were noted in the cardiac glands. The most interesting findings in this animal were confined to the fundic and pyloric gland regions. In the gland region of the fundus the pathological areas were patchy in distribution being separated from one another by normal mucosa. There was a loss of the covering epithelium and various degrees of destruction of the secreting tubules. In some places only the neck of the gland would be gone whereas in other places the entire secreting tubule would have been destroyed. In between the individual tubules there was an increased amount of interstitial tissue. The result of this was a distortion of the normal closely packed, palisade-like arrangement of the fundus glands. These changes are illustrated in Figure 5.

The sections of the pyloric mucosa revealed a museum of gastritic changes. The picture was so complete that the destructive process could be traced from beginning to end. The earliest changes have been described under dog No. 5-4 and illustrated in Figures 1 and 2. The next stage consisted of a destruction of the gland neck epithelium while the fundus portion of the glands continued to present a normal appearance. At the same time the interstitial tissue appeared to be increased and infiltrated with round cells (Fig. 6).

In Figure 7 the destruction has progressed to involvement of the fundus portion of the glands. The glandular tissue that had been destroyed was replaced by a loose inflamed tissue infiltrated with leucocytes, lymphocytes, and plasma cells. In many areas only a vestige of the gland neck was to be seen and the fundus portion of the gland was beginning to succumb to the destructive process.

In the final stage of destruction as illustrated in Figure 8, there was a complete loss of all the glandular structures. In some regions there remained only a vestige of the secreting acini. The mucosa was now replaced by a loose, amorphous tissue, diffusely infiltrated with round cells and plasma cells.

In examining sections comparable to those here mentioned but stained with muchematein it was noted that despite the amount of destruction that had taken place as long as there were any cytoplasmic remnants of the specific cells they took on the characteristic, deep blue staining reaction.

In the sections taken from the duodenum and jejunum there was a marked duodenitis and jejunitis. In the liver the usual fatty changes were noted.

Biliary Fistula Dogs with Bile Feedings

Dog No. 3-4. Large female mongrel weighing 23.6 kilograms. Complete external biliary fistula was made. This was the first animal to have bile feedings. About one-half of the bile collected was fed back to the animal by means of a stomach tube. Between feedings she was permitted to eat at will. This plan, however, was not effective for after the first 3 days the animal refused all food and often had an emesis after the feeding of bile so that probably very little bile was retained. The condition of the animal became progressively worse and after the seventeenth day a purulent material replaced the secretion of bile. Animal was sacrificed on nineteenth day because of poor condition and biliary infection.

Autopsy. Dog weighed 20.0 kilograms. The peritoneal cavity was clean. The extrahepatic biliary passages were dilated, injected, and edematous. When opened into there escaped an appreciable quantity of frank pus. The liver was diffusely reddened and presented a mottled appearance. The stomach appeared normal. On the anterior wall of the duodenum about 1 centimeter from the pylorus was an ulcer 1 by ½ centimeter in diameter. The adjacent duodenal mucosa was diffusely reddened.

Microscopic examinations. No changes in gastric mucosa were noted. Essential findings were limited to ulceration in duodenum as illustrated in Figure 9. The base of the ulcer extended into the muscularis. Covering the base of the lesion was an amorphous mass of cellular débris. The adjacent submucosa was edematous and appeared friable. All layers of the duodenum revealed a round cell infiltration. The reddened mucosal areas adjacent to the ulcer revealed a low grade duodenitis. No noteworthy changes were demonstrable in the jejunum. Usual fatty changes were observed in the liver.

Dog No. 7-4. Large, young, tan, female mongrel weighing 22.7 kilograms. Complete external biliary fistula was made. In this animal the plan of feeding

was changed. Twice daily the total secretion of bile for the 12 hour period previous was fed back after being mixed with the laboratory stock diet. For the first thirteen days after operation the animal appeared perfectly normal. On the thirteenth day there was evidence of biliary obstruction. The following day the abdomen was explored and a constricting ring located just proximal to the end of cannula. The extrahepatic ducts above this point were distended with "white bile." The obstruction was released and drainage to the exterior was re-established. Convalescence was slow but progressive. In 5 days the animal pulled out the drainage tube and had to be re-operated upon to re-establish drainage. Convalescence was again slow but progressive. Two days later the tube was again pulled out so that the animal was again explored to re-establish drainage. After this operation, the condition was only fair, and in a few days became poor and the animal was sacrificed on the twenty-fourth day of biliary exclusion. The bile-food feedings were religiously carried out during this entire period.

Autopsy. Dog weighed 16.3 kilograms. The peritoneal cavity was filled with a moderate amount of turbid yellow fluid. The omentum was thickened, injected, and edematous. The serosa of entire gastro-intestinal tract was deeply injected. Multiple perihepatic abscesses were noted. The mucosa of the stomach, duodenum, and jejunum presented a normal glistening appearance.

Microscopic studies. No pathological alterations were noted in stomach, duodenum, and jejunum. Superficial portions of liver revealed evidence of perihepatitis. The usual fatty changes were noted in the other sections of the liver.

Dog No. 14-4. Medium sized, tan, female mongrel weighing 19.5 kilograms. Complete external biliary fistula was made. Bile-food feedings were given the same as in previous animal. The condition was good for first 8 days when the drainage tube was pulled out. Drainage was re-established by a second operation. Three days after second operation the drainage of bile was replaced by a foul-smelling seropurulent material. From this point on the bile was obtained from other healthy dogs and the bile-food feedings were continued. For 7 days, the animal improved remarkably. After this her course was progressively down hill so that she was sacrificed after 3 weeks of biliary exclusion.

Autopsy. Dog weighed 15.9 kilograms. A diffuse purulent peritonitis was noted. The liver contained three walnut sized abscesses filled with thick pus. It was adherent to the lesser curvature of the stomach.

Microscopic studies. No alterations were noted in the gastric, duodenal, or jejunal mucosa. Fatty changes were again seen in the liver. Sections of liver from abscessed areas showed marked hepatitis.

DISCUSSION OF OBSERVATIONS

As previously mentioned the purpose of this study was to see if the ulceration in animals with biliary exclusion could be prevented by feeding bile. From this work it is concluded that gastro-intestinal ulceration can be prevented in this experimental animal with biliary exclusion by the adequate feeding of fresh bile obtained from dogs.

Additional evidence supporting this contention is found in the incidental observations of other investigators working with some phase of biliary secretion. First is mentioned the work of Whipple (32, a) and his associates. As mentioned above these workers diverted the secretion of bile to the exterior by way of a gall bladder fistula. It was noted that the course of these animals was rapidly down hill and that the dogs soon died. As a result their first conclusion was that in order to make adequate studies on these animals it would be necessary to maintain them in as nearly a normal condition as possible. To attain this end they tried various feeding procedures and finally concluded that bile is essential in the life of these animals on a mixed diet.

More recently Puestow (27) made an experimental study of the discharge of bile into the duodenum. For the purposes of his study a number of animals were prepared surgically so that an isolated segment of the duodenum containing the orifice and intramural portion of the choledochus, was exposed on the abdominal wall. For the purposes of this paper it can be seen that his was simply another means of excluding all of the bile to the exterior. Following personal communications with this author (28) it was learned that several animals following these surgical procedures died rather suddenly. Necropsies revealed perforated peptic ulcers beyond the gastro-intestinal anastomosis. Other animals, however, remained in an apparently healthy condition for several years. "These latter animals were frequently observed to lick bile from the fistulas and evidently ingested quite a large quantity of it."

The other side of this problem deals with the microscopic study of tissues involved in the disease picture. In this connection contributions by Konjetzny (20) and his associates are repeatedly referred to for 2 reasons. First, these workers have pointed out what changes are to be looked for in peptic ulcer disease.

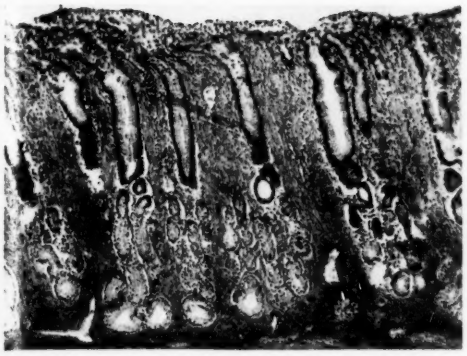

Fig. 1. Dog No. 5-4. Eight days of complete external drainage of bile. Section illustrates surface inflammatory exudate, loss of surface epithelium, beginning destruction of gland neck epithelium, and cyst cidilatation of outer margin of a gland neck. Hematoxylin and eosin. ×50.

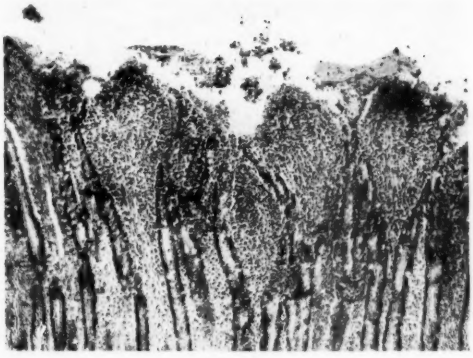

Fig. 2. Dog No. 5-4. Eight days of complete biliary exclusion. Hematoxylin and eosin stained section of first part of duodenum. This section illustrates surface exudate, loss of covering epithelium, and beginning erosion of tips of villi. ×50.

Second, a part of this problem has been not only to determine what associated pathological changes occur in these animals but to see if these findings are in any way comparable to the changes observed in man.

Gastritic changes in the pyloric region were described in dogs Nos. 4-4, 5-4, and 2-4. The question arises why these changes are not observed with equal frequency in all portions of the stomach? Though this question must remain unanswered at present, it is of interest that the same observation has been made for man by foreign investigators. It is only in the cases of a marked associated gastritis that changes can be observed elsewhere in the stomach i.e., in the fundus gland region. This fact applies equally well to this work as evidenced by the findings in dog No. 2-4.

Another characteristic of the associated gastritis as observed in man and in these animals is the patchy distribution of the pathological areas. The importance of this is best appreciated by referring to the studies in dogs Nos. 6-4 and 9-4. In these animals no alterations were noted in the gastric mucosa. This does not mean that such changes were not present but that the histological studies were inadequate. Either the pieces of tissue chosen were not large enough or were not selected from the proper regions of the mucosa.

Turning now to the alterations actually observed in these studies, one is immediately impressed with the marked similarity existing between these changes and the changes as observed in man. From the studies in dogs Nos. 4-4, 5-4, and the less involved regions in dog No. 2-4, it was noted that the destruction of the surface and neck epithelium of the pyloric glands constituted the earliest type of alteration in these glandular structures. Konjetzny has also described the gland neck epithelium as being first involved. In man, however, the changes seem to occur less rapidly. Here the first manifestation is often a hyperchromotosis of the nuclei. Shortly thereafter the lumen of the gland necks becomes packed or plugged with an exudative mass, which subsequently may be followed by a loss or destruction of the gland neck epithelium.

Mention of this obstructing mass of exudative cells in the necks of these glands leads us to the next type of change to be noted, namely, the cystic dilatation of some of the glandular structures. As will be recalled, this was noted in the pyloric gland necks of dog No. 5-4 and in Brunner's glands of the duodenum in dog No. 6-4. The factor responsible for the dilatation of these glands is still unknown but a number of explanations have been offered. It has been suggested that the exudative mass accumulating in the necks of these glands causes an obstruction at the outlet of the glands and in turn is responsible for the dilatations noted. Another explana-

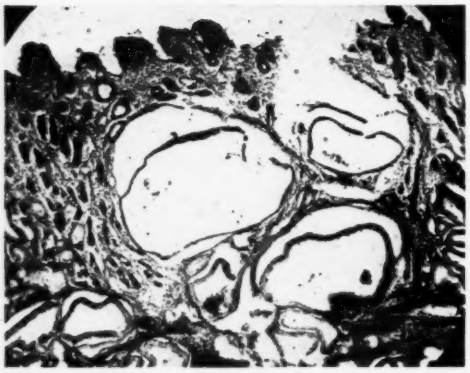

Fig. 3. Dog No. 6-4. Complete biliary exclusion for 13 days. Hematoxylin and eosin stained section of first part of duodenum. Illustrating complete loss of intestinal villi, almost complete erosion of tubular glands, and dilatation of secreting acini and tubules of Brunner's glands. ×50.

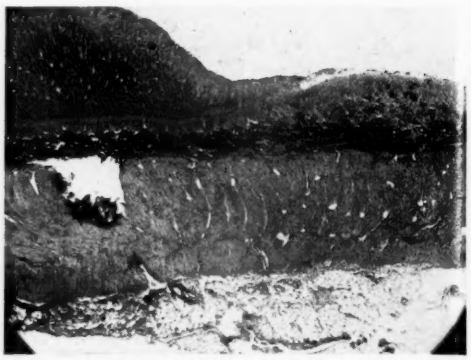

Fig. 4. Dog No. 9-4. Fifteen days of external drainage of bile. Hematoxylin and eosin stained section. Section illustrates active ulceration extending down to bulging edematous submucosa. Base of lesion filled with cellular débris. ×9.

tion offered is that when the normal glandular structures undergo atrophy or are lost, they are replaced by a new connective tissue. The new connective tissue laid down subsequently contracts and causes a stenosis or obliteration of the gland neck lumen.

Before dismissing the gastritic changes, attention must be called to the alterations noted in the fundus mucosa of dog No. 2-4. In this connection there was noted an increase in the amount of interstitial tissue resulting in a forcible separation of the closely packed gland tubules. There was also a destruction or atrophy of the glandular cells manifested first in the necks of the glands and then in the fundus portions. Further, in the regions where the process had progressed sufficiently to destroy entire tubules, there often could be seen one or more nests of from one to six parietal cells lying as vestiges in the path of the destructive process. These cells presented intact cell outlines and perfectly normal appearing nuclei.

This is of interest because Konjetzny has made identical observations in his studies of gastritis in man. To explain this phenomena, Konjetzny has attributed to the parietal cells a greater power of resistance to the destructive process which is responsible for these various alterations.

To introduce properly a discussion of the changes observed in the duodena of these animals, reference is made to a recent outstanding contribution of Puhl. This investigator made a detailed study of the development of gastric and duodenal ulcers in freshly resected human material. From these studies he has shown conclusively that the earliest stage of any ulcer begins in a minute erosion of the mucous membrane. He has also shown that by progressive development this minute erosion ultimately develops into the chronic, indurated lesion recognized clinically and pathologically as a peptic ulcer.

In this work it has been possible to trace an almost identical development of the ulcerous lesion. To relate more specifically the earliest stage or beginning of this lesion is manifested in dogs Nos. 4-4 and 5-4. In the sections taken from these animals there is chiefly a loss of covering epithelium of the villi. In some areas there is seen in addition a beginning erosion of the tips of the intestinal villi. In dog No. 6-4 this has progressed further to the point where in some areas the villi have completely disappeared and the erosive process is beginning to invade or already has invaded the layer of gland tubules. In animal No. 9-4 the glandular layer has been almost completely lost and the base of the lesion is now identified in close approximation to the muscularis mucosæ. In dog No. 3-4 the lesion has progressed beyond the muscularis mucosæ well into the submucosal layer and now is

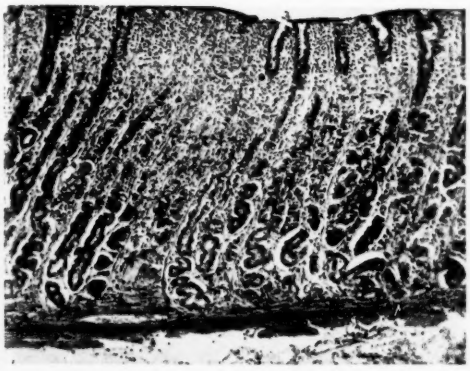

Fig. 5. Dog No. 2-4. Twenty-three days of complete external drainage of bile. Hematoxylin and eosin stained section of fundus glands. This section illustrates loss of surface epithelium, destructive changes in the tubules, and increased amount of interstitial tissue. ×50.

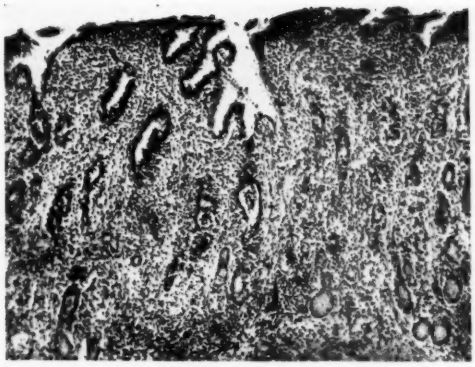

Fig. 6. Dog No. 2-4. Complete biliary exclusion for 23 days. Hematoxylin and eosin stained section of pylorus. Illustrates loss of covering epithelium, destruction of gland neck epithelium with fairly well preserved fundus secreting acini. ×50.

identified histologically as a peptic ulcer of the duodenum.

Up to this point little mention has been made of the conclusions derived from the study of the sections stained in the muchematein solution. It will be recalled that in the protocols of these animals, attention was repeatedly directed to the fact that in the case of the mucus secreting cells as long as any portion of the cell cytoplasm remained it continued to give the characteristic staining reaction. This is histological confirmation of the work of Whitlow that mucus laden and mucus covered cells are more resistant to acid-pepsin digestion than other cells. What appears to be more important, however, is the

Fig. 7. Dog. No. 2-4. Twenty-three days of biliary exclusion. Hematoxylin and eosin stained section of pylorus. Section illustrates complete loss of surface and gland epithelium. Fundus portion of glands is undergoing destruction and in some areas the glands have already begun to disappear. ×50.

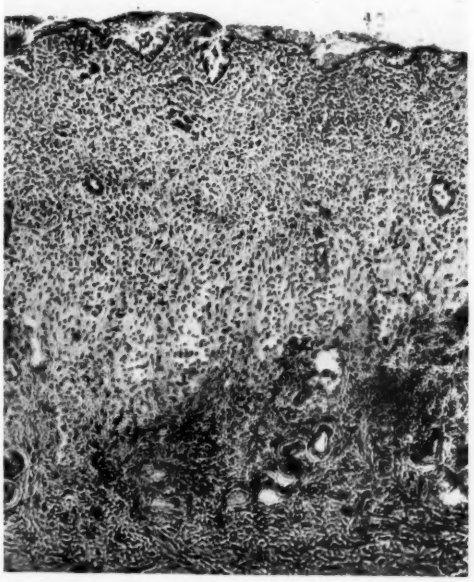

Fig. 8. Dog No. 2-4. Hematoxylin and eosin stained section of pylorus. This section illustrates an almost complete loss of all the glandular structures. The mucosa has been replaced by a loose tissue densely infiltrated with round cells and resembling that which is found in inflamed areas. ×68.

Fig. 9. Dog No. 3-4. Nineteen days of biliary exclusion with bile feedings. Duodenal ulcer stained with hematoxylin and eosin. Illustrates active lesion extending almost down to muscularis. Covering the base is an amorphous mass of débris. ×9.

fact that, although these cells possess a natural power of protection, they finally succumb to the influences of the destructive process.

In concluding, when a perspective view is taken of the changes occurring on both sides of the pyloric ring there remains another factor attracting attention. On the stomach side the changes are progressive and constitute chiefly a loss or atrophy of the gland structures. The mucosa, however, remains intact despite the fact that it becomes poorer in glandular elements. To describe this condition there has been coined the term "atrophic gastritis." Paralleling these alterations there are equally progressive changes occurring simultaneously on the intestinal side of the pyloric ring. Here the changes are manifested by a continual loss of tissue as described under the development of duodenal ulcers. At first the contrast in these changes may not seem important but, to the writer, this fact affords an explanation why there is a preponderance of the duodenal lesion over the gastric lesion in peptic ulcer disease.

SUMMARY

The purpose of this study was to show that in dogs the loss of bile was not only of paramount importance in inducing experimental ulceration but that by the adequate feeding of fresh bile from dogs these changes could be prevented.

Two of the 10 animals were sacrificed for control studies. In 8 dogs there was effected a complete external drainage of bile. Five were fed only the laboratory stock diet while the 3 remaining were fed the stock diet plus the bile that had been collected externally. In the dogs without bile feedings there developed not only peptic ulceration but the associated pathological changes of gastritis, duodenitis, and jejunitis. By incorporating fresh bile from dogs in the feedings, both peptic ulcers and the associated pathological changes could be prevented.

In the animals without bile feedings the associated changes of gastritis, duodenitis, and jejunitis were comparable histologically to the gastritis, duodenitis, and jejunitis associated with peptic ulcer disease in man.

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TISSUE DIAGNOSIS DURING OPERATION

Reliability of Terry's Supravital Technique in 1030 Biopsies

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THE committee on the treatment of malignant diseases of the American College of Surgeons endorses the rapid microscopic methods of tumor diagnosis, with the following advice: "In order that in patients with cancer the possibility of cure shall not be jeopardized, an exploratory operation should be conducted only under such conditions that the appropriate treatment, whether by surgery or by radiation, may be carried out immediately upon the establishment of the diagnosis by the pathologist by means of frozen sections."

It was not until Wilson in 1905 brought out his method of sectioning fresh tissue with the freezing microtome and staining it with polychrome methylene blue that tissue diagnosis during operation became practicable. This method has been tried out in the Mayo Clinic during the last 25 years on more than 28,000 malignant tumors. Besides Wilson and MacCarty of the Mayo Clinic, Bloodgood deserves the greatest credit for introducing the microscopic diagnosis from rapid sections as a routine procedure. Bloodgood states that such a diagnosis is at least equal in accuracy to that made later from carefully prepared paraffin sections.

In appreciating the advantages of rapid sections, some authoritative pathologists have been as reserved as the surgeons have been enthusiastic. Having made more errors by the use of frozen sections in cases of breast cancer than by the gross examination, Ewing has not resorted to frozen sections, in this field, for many years. Reimann said that the "very quick, five minute fix, cut and stain diagnosis" is looked on with suspicion by every good pathologist. According to Sternberg, of Vienna, the rapid method is often unreliable, especially in borderline cases in which an exact microscopic diagnosis would be essential during operation. Also in Warthin's and Dietrich's opinion, immediate microscopic diagnosis during operation has a very limited field.

In the controversy regarding the usefulness of tissue diagnosis during operation, any general acceptance or condemnation cannot be made. In any method of diagnosis, the success depends upon familiarity with a given procedure and the experience of the observer. The morphological diagnosis of a tumor is occasionally very difficult whatever the method of preparation, and long study may be necessary before the final classification of the growth is possible. The pathologist must have the courage, in cases in which the rapid method does not permit a clearcut diagnosis, to admit his failure. Then no surgeon will be misled.

The present custom, less common in this country than in Europe, of burdening a young surgical assistant with the responsibility of diagnosing tissue sections, will never permit a high standard of microscopic diagnosis during operation. The surgical assistant is obviously more interested in the technical side of surgery and leaves the rotating service in the laboratory before he has acquired a wide experience which is indispensable, especially for the diagnosis of frozen sections.

TECHNIQUE

The favorite rapid method used in German surgical clinics is that of Walz. After the tissue is fixed in hot formalin for 1 minute frozen sections are cut and stained with hematoxylin and eosin. For immediate diagnosis, during operation, the staining, dehydrating, and clearing have to be hurried so that frequently very unsatisfactory pictures are obtained. In my experience, these poorly differentiated and distorted sections, cannot compare with the beautifully stained preparations made by Wilson's supravital technique. The latter method enables one to see the intact cell, uninjured by boiling and

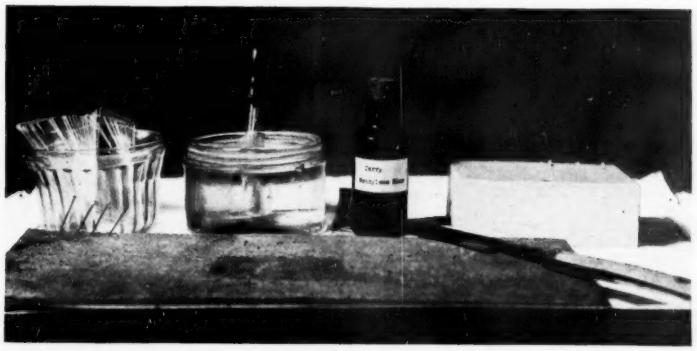


Fig. 1.

Fig. 2.

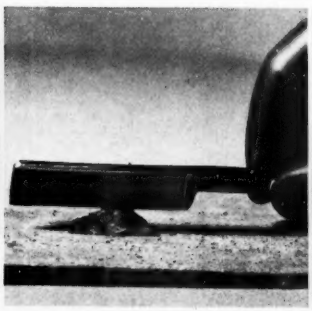
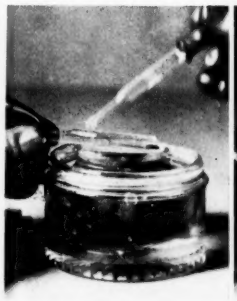


Fig. 3.

Fig. 4.



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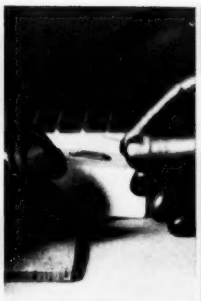

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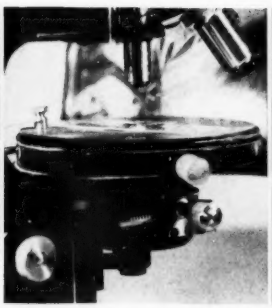

Fig. 7.

Fig. 8.

Fig. 1. The equipment consists of a sharp biconcave razor, a cork plate, pins, a water tumbler, a bottle of polychrome methylene blue, medicine dropper, glass slides, and cover glasses.

Fig. 2. Pin selected piece of tissue to cork plate.

Fig 3. Cut with razor a plane parallel slice from the wet tissue.

Fig. 4. Stain razor section superficially with methylene blue, for 1 to 3 seconds.

Fig. 5. Wash off carefully the excess of stain with tap water.

Fig. 6. Cover the stained surface firmly with a cover glass.

Fig. 7. Examine by transmitted light, using a 60 watt Mazda bulb. Arc light is necessary to transilluminate bloody tissue.

Fig. 8. After examination, preserve razor section in 10 per cent formalin for subsequent embedding in paraffin.

dehydration, while the cells in Walz's sections are often as shrunken and as different from the living cell as the raisin from the grape.

After I had used the method of Wilson for 5 years, my attention was called in 1929 to another supravital procedure which was devised by Terry. With increasing satisfaction and confidence, I came more and more to rely upon this method and, for several years, I have used it exclusively for the tissue diagnosis during operation. The entirely new principle of Terry's sections is that instead of having to cut tissue very thin to get histological detail, relatively thick sections are made with a biconcave razor and stained only on one side superficially with Terry's neutral polychrome methylene blue. The slice of moist tissue with the stained surface uppermost, is examined with artificial, transmitted light. The resulting microscopic picture is one of unexpected beauty and minute detail. Since only the uppermost cell layer is stained and the light is transmitted through the thick unstained part of the tissue slice, one has the impression of seeing a perfectly thin microtome section. The nuclei are stained a deep blue while connective tissue and muscle fibers are of a light rose color. High, as well as low, powers of the microscope may be easily employed. Few artefacts are encountered, since freezing, fixing, boiling or dehydrating are completely avoided. Many of the cells are still alive, and they are studied as nearly as possible as they are in the living body.

The microscopic picture remains for about 6 minutes, then the stain fades slowly. Sections may be restained or, if more material is available, a new section may be cut.

The three following reasons persuaded me to accept Terry's razor sections as a routine method for tissue diagnosis during operation.

1. Terry's method avoids the greatest disadvantage of the frozen section, namely, that the available tissue, when the biopsy specimen is very small, is used up by the rapid method and the employment subsequently of other methods of diagnosis is impossible. The preliminary cutting of razor sections does not exclude the later use of paraffin embedding. Even thin razor sections are usually thick enough to be cut in paraffin or celloidin.

TABLE I.—COMPARISON OF MICROSCOPIC DIAGNOSES OBTAINED FROM TERRY'S SUPRAVITAL SECTIONS AND PARAFFIN SECTIONS

Organ	Complete agreement between Terry's sections and paraffin sections	Agreement in regard to malignancy and benignancy	Disagreement
Skin	106	13	7
Oral cavity	65	3	3
Breast	124		
Respiratory tract	12	1	
Digestive tract	17		
Rectum	49		
Testis and ovary	42	1	
Cervix uteri	171	1	5
Fundus uteri	172	13	5
Prostate	59		3
Nervous system	10	1	
Bones and joints	30		1
Lymphatic system	49	2	7
Miscellaneous	53	1	4
Total 1030	959	36	35
Percentage	93.1	3.5	3.4

Moreover, the staining of sections with polychrome methylene blue does not prevent the subsequent staining of these with other stains, for the methylene blue is extracted completely when the tissues are run through alcohol. The objections brought out against Terry's method that it does not furnish permanent sections are without weight, because it should be the rule to follow the rapid method in every case with paraffin sections.

2. The second great advantage of Terry's method is the fact that it is so simple that the pathologist does not need a technician for the preparation of the section. The success of the method depends to a great deal upon this independence. Examination of the fresh surgical specimen by sight and touch, selection of the block, the cutting sensation with the razor, the avidity of the tissue for the stain, all these steps of preparing the slide often furnish immediate and essential information to the pathologist which a ready-made microscopic section handed by the technician may never reveal.

3. In rapidity, Terry's method is unsurpassed by any other histological procedure.

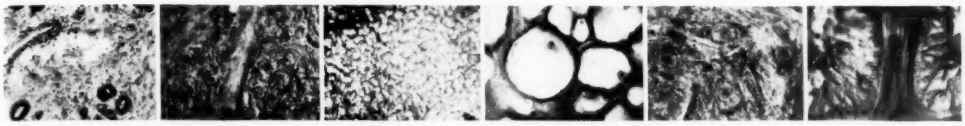

Fig. 9. Fig. 10. Fig. 11. Fig. 12. Fig. 13. Fig. 14.

Figs. 9 to 14.* Photomicrographs of Terry's sections.
Fig. 9. Chronic cystic mastitis.
Fig. 10. Solid carcinoma of breast.
Fig. 11. Lipoma (it is impossible to make frozen sections of fatty tissue but easy to make razor section).
Fig. 12. Folliculoma of ovary.
Fig. 13. Squamous cell carcinoma with horn pearls.
Fig. 14. Adenocarcinoma of uterus.

In less than 1 minute the tissue is cut and stained. Therefore, the study of several sections from different parts of the biopsy specimen is possible during the operation. Without causing delay for the surgeon, it is easy not only to diagnose the local lesion, but most accurately to determine the margin that should be given for a benign and for a malignant tumor and to ascertain whether glands near the local lesion which are exposed at the operation show metastases. Histological control of the whole tumor operation is made practicable by this ingenious method.

RESULTS

Terry's method has proved itself reliable during the last 5 years, not alone in the hands of the originator. The diagnoses made by Terry on razor sections of 2,000 tissues agreed in 96 per cent of the cases with those obtained by the pathologists of the Mayo Clinic using microtomes. Christeller, the German master of the microscopic art, was so impressed by Terry's method that he believed that it opened a new era in the field of biopsy. In 104 surgical specimens, including 40 malignant neoplasms, only three times was it impossible for him to make a correct diagnosis, because of the unusual structure of the tumors which required special staining methods.

*Figs. 9-14 represent a stage in the development of a photographic technique which will bring about great improvement. Unsatisfactory as these photographs are at this time, they cannot give any impression of the beauty in color and the detail in structure presented by the razor sections.

The difficulty that the surface of the razor sections is relatively uneven is easily and almost subconsciously overcome by the microscopist by turning the micrometer screw; while in photomicrography a large absolutely flat field is indispensable. The Leica or Zeiss-Contax Camera seems to be the only suitable apparatus for this special purpose, because these cameras allow instantaneous photomicrography during observation through a second eyepiece.

The perfection of photographing these supravital preparations is imperative, because only then it will be possible to have permanent records of these sections.

An excellent impression of the cell structure in supravital preparations is given by photomicrograph 7 which represents living cells of ovarian cancer, stained with Terry's methylene blue.

In 1932, I reported my first experiences with Terry's sections in a series of 368 biopsies, they allowed the same histological diagnoses as paraffin sections, in 94 per cent. In 98 per cent both microscopic diagnoses were identical in regard to malignancy and benignancy.

The results obtained in my recent series of 1,030 biopsies, including 303 malignant tumors, are recorded in Table I. Complete agreement between histological diagnoses obtained by razor and paraffin sections was found in 93.1 per cent, while malignancy and benignancy was recognized correctly in 96.6 per cent of the razor sections.

Terry's statement that the easiest organ to diagnose is the breast is substantiated by my own experience. There was not a single disagreement between razor and paraffin sections in this field. Much more difficult to diagnose were small friable uterine scrapings and small papillomatous tumors of the bladder, and the hardest tissues to diagnose were, in my series,

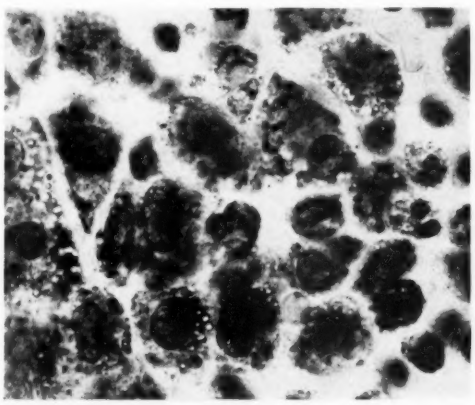

Fig. 15. Terry stained cells of ovarian cancer. The nuclear and cytoplasmatic structure is brought out perfectly by the supravital stain.

non-carcinomatous lesions of the lymph nodes. In 4 instances Hodgkin's disease was erroneously diagnosed by razor sections, as tuberculosis. On the other hand, carcinomatous metastases in lymph nodes were as easily recognized as the primary tumor.

In some tumors it was necessary to cut several sections before a diagnosis could be made. Very few razor sections were impossible of a diagnosis. In only 1.6 per cent of the biopsies was the surgeon informed that no definite diagnosis could be made by the rapid method. After removal of the tumor, the wound was closed and the radical operation was not considered, until a final histological diagnosis was obtained by means of paraffin sections. In these rare doubtful cases, it is obviously more in the interest of the patient to wait 24 hours for the preparation of paraffin sections than to proceed with a radical operation, on suspicion only.

Malignancy was not recognized in 5 cancer cases from razor sections. Two of these cases, however, represented so called early cancer of the cervix, and 1 was a small adenocarcinoma in a hyperplastic endometrium. In the 2 other instances the malignant changes were so minute that they were discovered only after studying many paraffin sections. These cases had metastatic cancer in lymph spaces of the neck and mesentery, respectively.

Up to the present, no false diagnosis of malignancy in a benign lesion has been given, except in a case of cellular angioma of the skin. From the razor section of the very bloody tissue I made the diagnosis of sarcoma. At this time, I had not yet learned that hemorrhagic tissue can be easily transilluminated by using arc light in place of the 60 watt Mazda lamp which is satisfactory for most razor sections. Also in this case, no harm resulted to the patient from the erroneous diagnosis, because the surgeon performed only a local excision of the tumor.

SUMMARY AND CONCLUSIONS

Terry's supravital technique has been adopted as the most favored routine method of diagnosing tumor tissue during operation. Its reliability equals that of frozen sections.

In rapidity, it is not surpassed by any other histological procedure, and it permits, therefore, the microscopic control of a whole tumor operation without causing any delay to the surgeon.

It does not prevent subsequent employment of paraffin or celloidin sections, even if the biopsy specimens are of extremely small size.

It is noiseless and can be employed in the operating room without elaborate equipment.

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CLINICAL SURGERY

FROM THE SURGICAL SERVICE OF THE ROOSEVELT HOSPITAL

A METHOD OF CLOSURE OF TEMPORARY EXTERNAL FECAL FISTULA

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FOR the past five years we have employed a method for the closure of temporary fecal fistulas which has been satisfactory. It has been used principally for the closure of temporary cecostomies and colostomies; however, it is equally applicable to small bowel fistulas. It is described for the reason that the operator for an occasional fecal stoma often wishes to know explicitly the steps of a relatively safe procedure, and it is quite infrequent that textbooks, monographs, or articles on this phase of intestinal surgery give a minute account. We claim no originality for the procedure, many others no doubt have followed the various steps in exactly the same manner. Usually

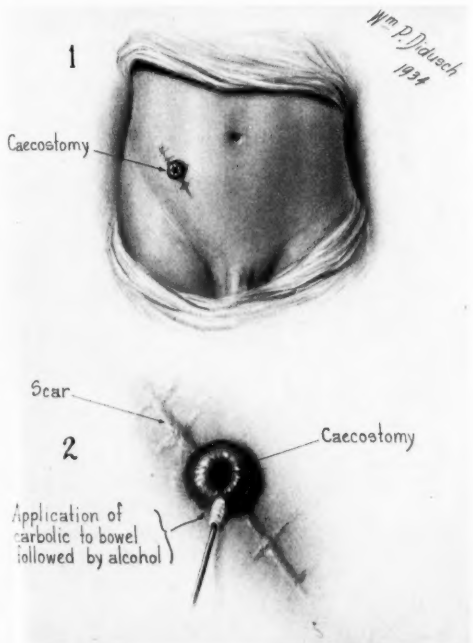

Fig. 1. *1*, A pouting cecostomy stoma with the intermuscular incision completely healed above and below. *2*. Phenol being applied in an attempt to sterilize the infected mucous membrane.

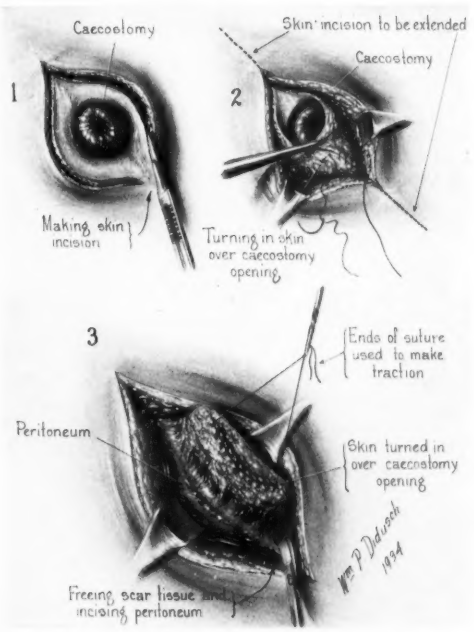

Fig. 2. *1*, An elliptical incision in the skin is made one inch away from the fistulous opening. *2*, The flaps of skin are carefully sutured over the opening with a continuous suture of medium sized silk; and the skin incision is then extended above and below. *3*, The ends of the continuous silk sutures are utilized for the purpose of traction, carefully but gradually pulling the closed in stoma out 2 inches above the level of the surrounding skin. The muscular layer is separated and pushed away and when necessary the peritoneal cavity can at this stage be opened into.

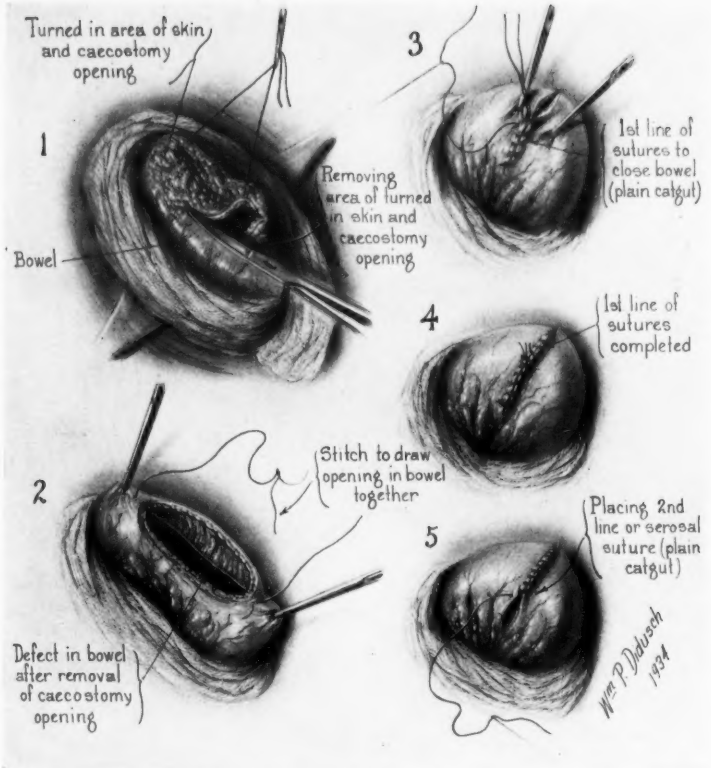
Turned in area of skin and caecostomy opening
1
Bowel
Removing area of turned in skin and caecostomy opening
3
1st line of sutures to close bowel (plain catgut)
4
1st line of sutures completed
2
Stitch to draw opening in bowel together
5
Placing 2nd line or serosal suture (plain catgut)
Defect in bowel after removal of caecostomy opening
Wm P Didusch 1934

Fig. 3. *1*, Gauze pads are packed around the bowel to protect the peritoneal cavity from becoming soiled. The turned in stoma is then excised and healthy bowel wall exposed made thus ready for suture. *2*, The opening into the lumen of the bowel and a suture placed in the center of the proposed line of closure. *3*, The defect is closed transversely to the long axis of bowel wall. *4*, First layer of a continuous suture shown completed. *5*, A second tier, a serosal suture is made, continuous plain catgut being used.

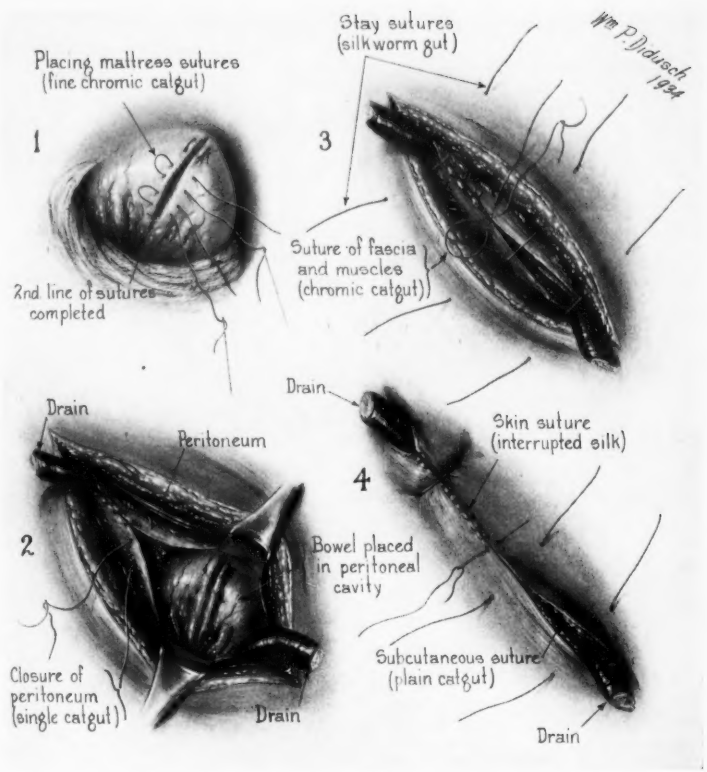

Fig. 4. *1*, Reinforced mattress sutures of fine chromic catgut are placed forming a third tier of sutures. *2*, The closed bowel dropped back inside the peritoneal cavity. Small drains are placed in either angle of the wound and the peritoneum closed. *3*, Stay sutures are placed and the muscles and fascia closed with figure-of-8 fine chromic catgut sutures. *4*, Fine plain catgut is used to bring together the subcutaneous tissue, silk to skin, stay sutures being tied and end of drains protruding.

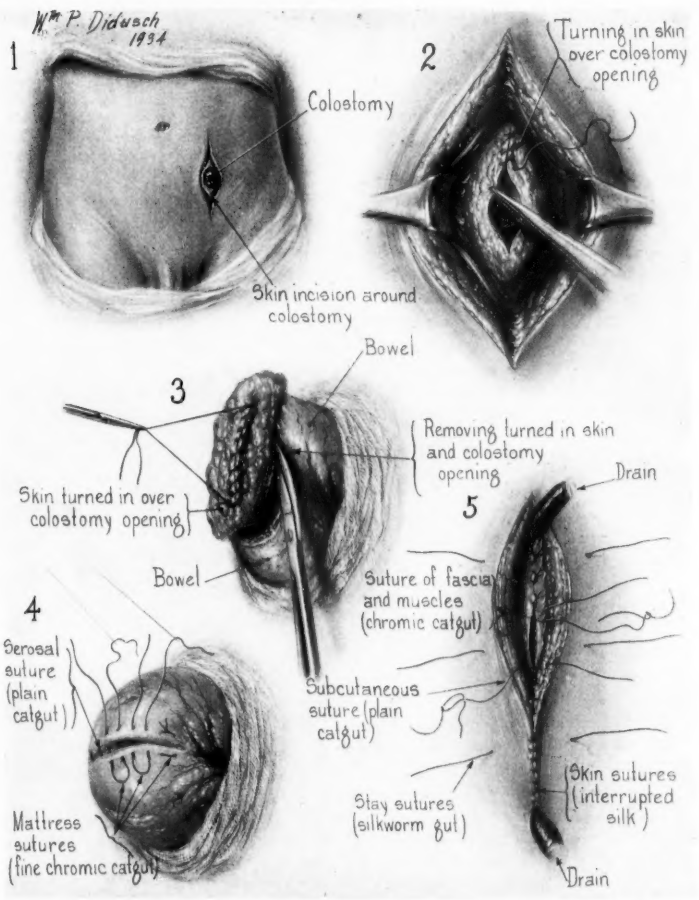

Fig. 5. Shows the various similar steps in the closure of a left sided colostomy temporary fistulous opening.

it is an intraperitoneal method. Yet we have on numerous occasions been able to close extraperitoneally, particularly if there is present a moderately thick layer of extraperitoneal fat, for as traction is made on the temporarily closed stoma there is more give to the bowel loops themselves, due to elasticity of the adherent extraperitoneal fat layer. We have had but moderate fear of peritonitis, for we, like others, believe there exists a fair local immunity due to vaccination from the previous operative maneuver and the functioning stoma with its fecal flow. It is not an aseptic method, none that I know of are, yet care is used to minimize unnecessary soiling.

Before an attempt is made to close a fecal fistula, it is obviously necessary to know that there is no obstruction present below in the intestinal tract. A practical pre-operative measure, occasionally overlooked, is an enema or a colonic irrigation to clear away inspissated fecal matter; one can well imagine impacted material in the rectum spelling failure to an otherwise satisfactory closure.

A thorough 3 day pre-operative preparation will add considerably not only to the ease and neatness with which the procedure is carried out, but also to the comfort of the patient after operation. Furthermore, a supposed simple procedure

of closure of the colostomy may easily and frequently become a major surgical undertaking; for instance, a resection may be required and this would tax the vitality and resistance of a well individual. The pre-operative preparation consists of a low residue diet for 3 days before operation, morphine sulphate, 1/6 grain every 4 hours by the clock during the 36 hours just prior to operation, and 1,000 cubic centimeters 5 per cent glucose twice daily for 2 days before the day of operation. In patients with a cecal stoma, often the skin surface about the opening is excoriated from the pouring forth of the contents of the small intestine. We have found aluminum powder (the silver-like dressing for radiators) a most excellent protection against this painful and sometimes hazardous fecal irritant. A non-excoriated, healthy skin about the fistulous opening means much toward a rapidly and completely healed wound.

I have used this procedure twenty-eight times and in no instance has there been the necessity of a second operation. In three there occurred a "spitting out" of flatus and a small amount of fecal matter for several days followed by spontaneous closure.

The accompanying illustrations with the descriptive notes below seem sufficiently clear to obviate the necessity of any further explanation of the operative procedure.

SURGICAL TREATMENT OF JOINT TUBERCULOSIS

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THE controversy aroused by the subject of the proper treatment of joint tuberculosis obscures the main issue. The heliotherapists proclaim triumphs and the surgeons show their excellent results but each is singularly silent about those patients under his care who fail to respond to treatment.

After one has seen patients with joint tuberculosis treated for 3 to 5 years with heliotherapy and the disease still remains active, or when one has seen patients with various infected joints, solidly fused surgically, die of tuberculosis after a period of years, the question must be raised whether either of these groups has squarely faced this important problem. The excellent results are always a pleasure but the poor results in this type of work must relentlessly be studied if we are to learn by our failures.

I believe that joint tuberculosis as a distinct entity does not exist and should cease to be considered as such. The patients with manifestations of tuberculosis in the joints must be considered as cases of tuberculosis with a primary focus in the lungs, lymph glands, tonsils, etc., in whom the joint has been secondarily invaded. To consider such a patient solely in terms of the invaded joint is, of course, erroneous. The condition of the lungs, upper respiratory tract, gastro-intestinal tract, and genito-urinary tract is of paramount importance in these patients who must be studied as a whole in relation to their tuberculous infection. Until each series of patients reported with joint tuberculosis is so studied, we shall not, I believe, arrive at any exact knowledge of the problem of their treatment or prognosis.

During the years 1924 to 1931, inclusive, at Sea View Hospital, there were 12,386 admissions, all presumably with tuberculosis. A careful study of the records show that among these 12,386 admissions, there were 500 cases of joint tuberculosis, an incidence of about 4 per cent. Many cases of suppurative arthritis and osteomyelitis, a few cases of Charcot's disease of knee, hip, and spine, and many cases of chronic arthritis have all been weeded out.

Joint tuberculosis is usually a true joint manifestation, starting in the synovial tissue, and present for a long time before the bone is grossly involved. Tuberculosis of the shaft of the long bones is relatively rare. We have seen the radius involved once, the femur once, the tibia once, the humerus once, and the os calcis twice, all proved by tissue removed. There have been many cases of tuberculosis of tendon sheaths of hands and feet and a few fascial abscesses without bone foci.

From the 500 patients who came under the care of the orthopedic department during these years, we selected 210 patients as candidates for surgical treatment. The remainder were rejected as their general condition did not warrant surgical intervention. Of these an overwhelming majority died. In spite of this selection, those patients surgically treated, in most instances, would be considered a highly unfavorable group when it is noted that of the 210, 153 showed evidence of pulmonary tuberculosis. During the work with these patients, the salvage aspect of this type of surgery has become increasingly clearer to the author.

Race or nationality. The race or nationality of the patients comprising this group is extremely cosmopolitan. We have considered foreign born and the first generation in this country as belonging to the parent stock. Native Americans are far outnumbered by the foreign racial strains. Corroboration of known facts of racial immunity and susceptibility to tuberculosis is abundantly shown. The Jews show a high resistance to the disease while the West Indian negroes, American negroes, and Irish succumb in large numbers. West Indian negroes had a mortality of over 41 per cent; the American negroes a mortality of 29 per cent; the Irish a mortality of 26 per cent; native Americans 15.7 per cent; and the Jews 11.7 per cent (see Table I).

Joints involved which were treated surgically. This group of 210 patients had 224 joints involved which were treated surgically (see Table II). It will be seen that the weight bearing joints present the great bulk of the problem in joint tuberculosis.

Operations performed. Upon these 210 patients with 224 joints treated surgically, there were performed 261 operations (see Table III).

Diagnosis. Every effort has been made to establish a definite diagnosis. Clinical examination and roentgenographic picture of a tuberculous joint—marked atrophy, thin joint space, capsular distention, abscess formation, etc.—are pretty frank in the advanced case but, in the early case in which the diagnosis is infinitely more

TABLE I.—RACE OR NATIONALITY OF 210 PATIENTS IN RELATION TO MORTALITY

Race	Patients	Deaths	Mortality* per cent
American negroes	38	11	29
Irish	34	9	26.4
Italians	33	5	15
West Indians	24	10	41
Americans	19	3	15.7
Jews	17	2	11.6
Germans	15	3	20
Poles	5	4	*
Greeks	4	0	
Spaniards	3	2	
Hungarians	3	0	
English	2	1	
Scotch	2	2	
Canadians	2	1	
Lithuanian, Russian, Slovak, Finnish, Bohemian, Japanese, Bulgarian, Scandinavian, French, 1 each	9	6	
Total	210	59	

*Mortality not computed for groups under 10 in number

important, they are often slender props to lean upon. The clinical picture of spasm, atrophy, limp, and deformity applies equally to suppurative arthritis in many instances, so the diagnosis may well be uncertain until tissue or exudate is examined.

In 101 of the 224 different joints, the evidence was conclusive after tissue examination or guinea pig inoculation. In 110 cases the spine was involved and since in these cases it is impossible to get pathological corroboration unless an abscess can be aspirated or another joint is involved which may be explored, it will be appreciated that a very high percentage of the available joints were proved cases (see Table IV). If the spine cases were subtracted it will be seen that 87 of 113 available joints, or 76.5 per cent, were proved positive. In 14 spine cases it was possible to recover tubercle bacilli or tissue to add to the certainty of diagnosis.

TABLE II.—RELATIVE FREQUENCY OF INVASION OF THE VARIOUS JOINTS IN THIS SERIES

	No.	Per cent
Weight bearing joints		
Spines	110	49.3
Knees	44	19.7
Hips	25	11.2
Sacro-iliac joints	16	7.1
Tarsal joints	13	5.8
Ankle joints	4	1.8
Symphysis pubis	1	0.4
Total lower extremity or spine	213	95.3
Non weight bearing joints		
Shoulders	2	0.8
Elbows	5	2.3
Wrist or carpal joints	4	1.6
Total upper extremity	11	4.7

Principles of treatment. Our treatment has been carried out with the idea that each patient has systemic tuberculosis. We have often kept patients in bed for several months before operation and for 8 months to a year after operation. They have had as good food as possible and during the summer months all the sunlight that our climate affords. All of our children get cod liver oil. The operative treatment aims to eliminate the surgical focus by putting the joint or joints involved at complete rest, and the most effective means of accomplishing this is by operative fusion. If an extremity joint is overwhelmingly involved and any attempt to save it endangers the patient's life, amputation is resorted to.

The patient in relation to his tuberculosis. In considering these patients from the standpoint of their systemic tuberculosis, we have divided them into four main groups as follows:

A. Patients with pulmonary tuberculosis and positive sputum.

B. Patients with pulmonary tuberculosis and negative sputum, with metastatic spread to other organs.

C. Patients with pulmonary tuberculosis and negative sputum, with no metastatic spread to other organs.

D. Patients with evidence of no pulmonary tuberculosis.

The total number in each group and the mortality is given in Table V. It will be seen that among the 93 patients in groups A and B, there occurred 48 of the 59 deaths while the remaining 117 patients had only 11 deaths among them.

The group with the positive sputum and cavities in the lungs should, in all probability, be absolutely avoided as operative choice. Unfortunately, the second group with inactive pulmonary disease but with metastatic spread to other organs frequently can not be recognized prior to operation and will always furnish a large part of the mortality in any series of cases of joint tuberculosis. Here are the cases of tuberculosis of the genito-urinary tract, meninges, and peritoneum. We have seen these metastases develop too often in patients awaiting operation to believe that the operative procedure plays any part in the spread.

Follow-up examination. The follow-up in a group of patients who are notorious drifters is extremely difficult. In spite of this, our average

TABLE III.—OPERATIONS PERFORMED ON THESE 210 PATIENTS

	Operations	Total operations
I. Spines		
1. Spine fusion	109	
2. Spine fusion and laminectomy	9	
3. Exploration of spine for pseudarthrosis	9	
4. Laminectomy on fused spine (for paraplegia)	2	129
II. Knee joints		
1. Knee fusion	41	
2. Amputation	5	
3. Repair pseudarthrosis	4	
4. Biopsy	2	
5. Osteotomy of fusion for flexion	1	53
III. Hip joints		
1. Fusion	25	
2. Repair pseudarthrosis	2	
3. Repair second pseudarthrosis	1	
4. Biopsy	1	
5. Osteotomy for deformity	1	30
IV. Sacro-iliac joint		
1. Fusion	17	17
V. Tarsal joints		
1. Fusion	9	
2. Amputation	4	13
VI. Ankle joint		
1. Fusion	3	
2. Amputation	1	4
VII. Symphysis pubis		
1. Fusion	1	1
VIII. Upper extremity joints		
1. Fusion	11	
2. Repair pseudarthrosis	2	
3. Amputation	1	14
Total operations		261

TABLE IV.—INCIDENCE OF POSITIVE DIAGNOSIS

The diagnosis was proved by tissue examination or guinea pig inoculation in various joints as follows:

Joint	Total	Number proved positive
Spines	110	14
Knees	44	35
Hips	25	18
Sacro-iliac	16	13
Tarsal	13	7
Ankle	4	4
Symphysis pubis	1	0
Upper extremity	11	10
	224	101

final follow-up examination has been 3 years. We have not relied on correspondence but have seen and examined each patient.

End-results. Any attempt to gauge accurately the end-results in cases of joint tuberculosis is fraught with great difficulty. One has only to see non-operative patients who have had their disease light up after 30 years of quiescence or to have sent home patients with fused knees and hips in excellent condition and then several years later to see them die of tuberculous meningitis, to realize that we are dealing with an insidious disease of extreme chronicity. Each of these patients is in constant danger. Our operative treatment at best leaves them with stiffened joints which are better by far than the active disease, but there can be no restitution of function. I have rated these patients in three groups as far as results are concerned.

a. *Excellent*—those patients with solidly fused joints, with no active pulmonary disease, who can and have left the hospital to resume a relatively normal life.

b. *Uncertain*—those patients with solidly fused joints in most instances but with active pulmonary disease or amyloid disease from prolonged suppuration. Many of these patients have or have had positive sputum. Some are still in the hospital. Many have left the hospital but we have no feeling of security for their future.

c. *Died*—this is the only group that we can be absolutely certain about.

TUBERCULOSIS OF THE SPINE

In this series 113 patients had tuberculosis of the spine and 110 of these were treated surgically. In 85, the spine was the sole focus while in 25, there was an additional joint or joints involved. The number of male patients was 64; of females, 46. Five patients were under 5 years of age; the oldest was 62; average age at onset was about 23 years (see Table VI).

Duration of symptoms. The average duration of symptoms was 3 years and 2 months. The longest period was in a patient in whom the condition had persisted for 32 years prior to operation. In 7 the condition had persisted over 10 years. In 17 patients the disease had been present less than a year.

Diagnosis. The diagnosis was made by physical examination and roentgenogram. Backache or pain was generally complained of and there was almost invariably spasm, tenderness, and deformity. The roentgenogram showed a thin intervertebral space progressing to actual destruction of the vertebral body above and below with abscess formation. In 14 cases it was possible

TABLE V.—MORTALITY IN RELATION TO EXTENT OF SYSTEMIC TUBERCULOSIS INFECTION

	A Patients with pulmonary tuberculosis and positive sputum	B Patients with pulmonary tuberculosis and negative sputum, with metastatic spread to other organs	C Patients with pulmonary tuberculosis and negative sputum, with no metastatic spread to other organs	D Patients with evidence of no pulmonary tuberculosis
Entire group				
Patients	53	40	60	57
Deaths	24	24	4	7
Mortality per cent	47	60	6.6	12.2
Tuberculosis of spine				
Patients	25	22	35	28
Deaths	11	14	3	4
Mortality per cent	47	63	8.5	14
Tuberculosis of knee joint				
Patients	11	8	15	10
Deaths	4	4	0	1
Mortality per cent	36	50	0	10
Tuberculosis of hip joint				
Patients	7	3	7	8
Deaths	3	3	1	2
Mortality per cent	42.8	100	14	25
Tuberculosis of sacro-iliac joint				
Patients	5	7	2	2
Deaths	4	3	0	0
Mortality per cent	80	42.8	0	0
Tuberculosis of tarsal joints				
Patients	5	2	2	4
Deaths	3	0	0	0
Mortality per cent	60	0	0	0
Tuberculosis of upper extremity joints				
Patients	2	4	3	2
Deaths	0	1	0	0
Mortality per cent	0	25	0	0
Multiple foci group				
Patients	4	18	12	9
Deaths	3	9	0	2
Mortality per cent	75	50	0	22

to recover tubercle bacilli or tuberculous tissue from abscesses or additional joint foci, to add to the certainty of the diagnosis.

Areas of spine involved. These 110 spines, including additional spine foci in some instances were distributed as follows: 3 cervical; 4 cervicodorsal; 60 dorsal; 12 dorsolumbar; 30 lumbar; 5 lumbosacral.

An examination of the individual vertebræ involved shows the great bulk of the lesions in the lower dorsal vertebræ with the cervical spine infrequently involved, while the lumbar spine is quite often the site of the disease. In this series the apex of maximum involvement extends from the seventh dorsal to the twelfth dorsal (see Chart 1).

Paraplegia. Among the more important complications of tuberculosis of the spine is paraplegia. This was first noted and described clinically by Sir Percival Pott in a paper titled "Remarks on that kind of palsy of the lower limbs which is frequently found to accompany curvature of the spine and is supposed to be caused by it." London, 1779.

The spinal cord pressure is caused by an abscess and not by bony deformity. It is associated chiefly with disease of the dorsal spine. In this series, 33 patients, or 29 per cent of the total, showed these symptoms. The vertebræ involved extended from the first dorsal to the first lumbar with the maximum involvement at seventh, eighth, and ninth dorsals (Chart 2). Paraplegia may occur at any age (see Table VI).

The symptoms vary from mild spinal cord pressure with resulting stiffness, weakness, and awkward gait, increased activity of the reflexes, and impaired sensation of the lower extremities to complete spastic paralysis with anesthesia and loss of control of the anal or urinary sphincter or both. Bed sores develop only in the most severe cases and then usually as terminal manifestations.

Eight of these patients presented evidence of mild or imcomplete pressure, while the 25 remaining had complete paraplegia. The mild cases all cleared up spontaneously and, with one exception, occurred before any operative procedure had been instituted. This single exception was in a patient who 5 years after spine fusion showed weakness, mild spasticity, and sensory disturbance, which cleared up after a few months' rest in bed.

The 25 patients with evidence of serious spinal cord pressure presented a more complicated problem. Ten of them developed symptoms before operation and 15 afterward. In this group the symptoms cleared up in 13 patients, and in 12 there was no improvement; these latter all died of the disease at an average of 9 months after operation, a 48 per cent mortality. Those patients who failed to recover were for the most part in the groups in which failure to survive is frequent, i.e., those with positive sputum and those with negative sputum but metastatic spread to other organs.

Laminectomy was performed on 11 patients, of whom 6 recovered and 5 did not. This operation was at times done in conjunction with a fusion and at times subsequently. In each instance a definite block in pulsation of the spinal cord was noted. Tuberculous granulation tissue was often found over the dura, which was not opened.

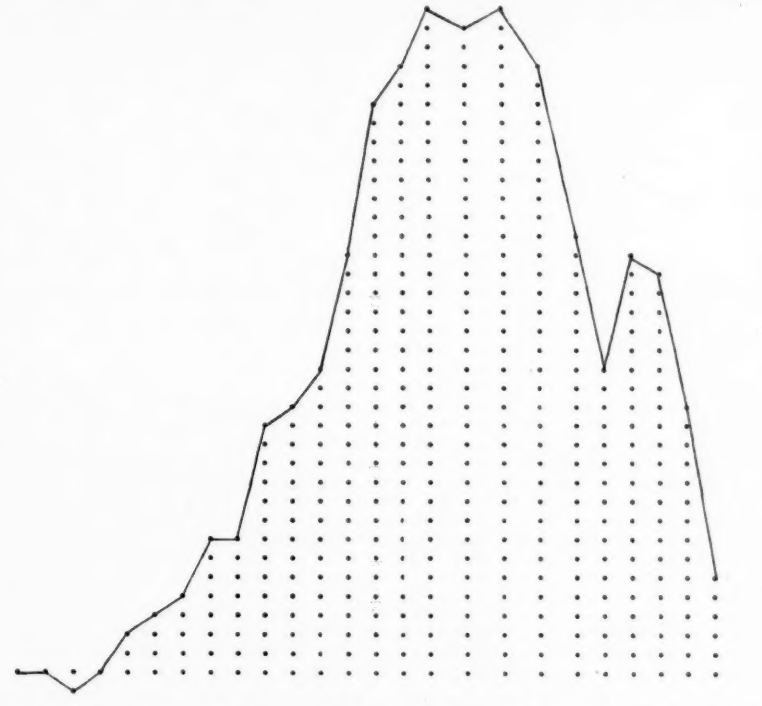

Chart 1. Vertebræ involved in this series of 110 patients.

There were several instances in which, following the operative release of pressure, the symptoms cleared up with dramatic speed. One child of 9, whose paraplegia had been complete for 4 months, had recovered almost entirely 2 weeks after operation.

Abscess. Either paravertebral or psoas abscess was noted in 75 of these 110 operative spine cases. Many of these were repeatedly aspirated successfully, thus preventing sinus formation. In healing the abscesses tended to calcify. Of the 75 patients with abscesses, 29 died, a mortality of 38.6 per cent.

Sinuses. Sinuses increase the gravity of the problem greatly. Amyloidosis after prolonged suppuration is too well known to require comment. Twenty-four patients had sinuses and 13 or 54 per cent died.

Treatment. In these 110 operative spines the surgical treatment has been spine fusion of the

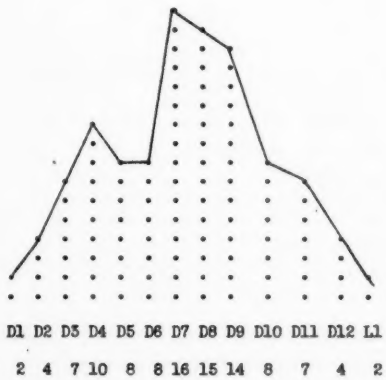

Chart 2. Vertebræ involved in those 33 patients developing paraplegia.

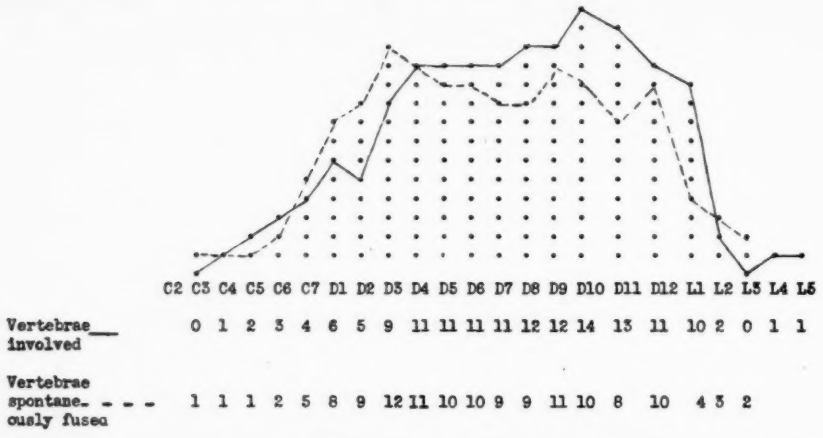

Chart 3. Spontaneous fusion, showing in 25 patients the relation between vertebræ involved in the disease and those spontaneously fused. The broken line designates the vertebræ spontaneously fused while the continuous line designates those involved by the disease. In some instances, such as at third cervical and from seventh dorsal to third dorsal at third lumbar those vertebræ spontaneously fused exceed in number those involved by the disease, which illustrates the mechanical nature of this attempted repair.

Hibbs type, extending from 2 sound vertebræ above and below. In a few cases the fusion has included only 1 sound vertebra above and below.

Spontaneous fusion. At operation spontaneous fusion was found in 25 patients. In 4 others there was noted a partial or incomplete fusion. The age of these patients with spontaneous fusion was distinctly young, 64 per cent of them being under 20 years of age (see Table VI). The average duration of symptoms was almost 6 years, which is twice that for the entire series of 110 patients with tuberculosis of the spine.

Nineteen of the 25 had a long, rather sharp kyphos involving an average of 8 vertebræ of which the average number spontaneously fused was 6. The 6 remaining had only a moderate kyphos, with an average of only 3 vertebræ spontaneously fused.

The longest spontaneous fusion was 12 vertebræ, and there was one instance of 11 vertebræ, and another of 10 vertebræ. Two patients had 2 separate areas of spontaneous fusion. In most instances, vertebræ above and below were operatively added to those spontaneously fused as the area was inadequate. If there were 2 separate areas of spontaneous fusion, these were connected and additional vertebræ were added above and below. In one patient with 11 vertebræ spontaneously fused, no vertebræ were added, as the fusion was considered mechanically adequate.

Chart 3 shows graphically the relation between those vertebræ involved in the disease and those spontaneously fused. It is interesting to note that, in some instances, the vertebræ spontaneously fused were outside the diseased area but were mechanically in the kyphos. This heroic effort of nature to remedy a bad mechanical situation in the spine that has arisen from destruction of vertebral bodies is, of course, the inspiration for our surgical treatment of this disease. Unfortunately, nature works slowly and with tremendous deformity. These patients who showed this reparative phenomenon of spontaneous fusion had had the disease for many years with severe destruction of vertebral bodies and great deformity for the most part. They are a group with greater resistance to the disease than most. Of the 25, but 5 died, 20 per cent mortality. These deaths were due to metastatic spread to other organs in 2 instances, to amyloid disease in 1, and the remaining 2 fatalities were cardiac deaths.

Failure of fusion or pseudo-arthrosis. We have had no braces available at Sea View Hospital and have not used plaster-of-paris jackets, so our patients have had no postoperative immobilization, except to lie flat on a fracture board. The question of solid bony fusion is of the utmost importance so we have carefully examined each patient and have had postoperative roentgenograms

TABLE VI.—AGE INCIDENCE IN VARIOUS FORMS OF JOINT TUBERCULOSIS

Years	Patients with tuberculosis of spine			Patients with tuberculosis of knee joint	Patients with tuberculosis of hip joint	Patients with tuberculosis of sacro-iliac joint	Patients with tuberculosis of tarsal joints	Patients with tuberculosis of upper extremity joints	Patients with multiple joint foci
	Entire group	With paraplegia	With spontaneous fusion of spine						
1–10	24	5	6	4	9	0	1	1	8
11–20	20	4	10	12	6	5	3	4	15
21–30	26	8	4	15	7	7	3	3	11
31–40	17	6	2	3	2	3	2	1	7
41–50	14	7	2	10	1	1	3	1	1
51–60	8	2	1	0	0	0	1	1	1
Over 60	1	1	0	0	0	0	0	0	0
Total	110	33	25	44	25	16	13	11	43

made at intervals. Though the fusion may seem solid clinically, it is very rare, except in children, that the roentgenograms show a satisfactorily calcified bony mass under 6 months. With the increasing kyphos, persisting pain and spasm, failure of fusion may be suspected and the roentgenogram may show it.

End-results of tuberculosis of spine. From the standpoint of operative technique, we have had occasion to doubt that fusion was solid in 15 cases. In 5 of these the operation was done at other hospitals; of these, 3 were of the Hibbs type and 2 were bone grafts of the Albee type. At operation one Hibbs fusion was found solid with adequate bone and the 2 others were repaired. The tibial bone grafts were too short, had fractured at the center, and were partly absorbed. These also were repaired. Of the 10 remaining cases which were originally operated upon at Sea View Hospital, 4 when explored were found to have solid, massive, bony fusion. Three of these were in children whose kyphos had definitely and markedly increased, apparently due to massive vertebral destruction and the final deformity was assumed after the diseased bodies settled into contact.

Of the 6 remaining suspected of failure of fusion, 1 patient would not permit a second operation. The 5 others were proved to have failure of fusion at the second operation and were repaired. These failures, except 1 in the dorsal spine, were at the dorsolumbar junction or upper lumbar spine, an area of great mobility and strain in which we might anticipate a certain number of failures. The incidence of only 5 proved cases of failure of fusion in 110, or 4.5 per cent, speaks highly for the effectiveness of the operative procedure. We have had ample opportunity to observe the effective massive fusions that occur in most instances. At postoperative laminectomy, we have found this mass of bone as thick as five-eighths of an inch covering the laminæ and forming an entirely adequate brace.

The end-result rating of these 110 patients will be seen in Table VII.

In analyzing cases of tuberculosis of the spine in relation to their systemic tuberculosis, we find practically 80 per cent of the mortality lies among those patients with positive sputum or metastatic spread to other organs (see Table V).

Of the 32 who died with tuberculosis of the spine, 29 had abscesses and 13 had sinuses. Deaths in the more favorable groups were due to amyloid disease and in one or two instances to cardiac disease.

In analyzing these deaths in tuberculosis of the spine in relation to age, we find that the mortality in the first decade is low and that it reaches its maximum in the third decade (see Table IX).

An analysis of the deaths in relation to the area of the spine involved shows that there is a pretty even distribution with the lumbar spine showing a slightly higher mortality rate (see Table VIII).

TUBERCULOSIS OF THE KNEE JOINT

In our experience tuberculosis of the knee joint is the most favorable site for surgical treatment. The joint is easily explored for diagnostic purposes and is not difficult to fuse.

In this series, there were 44 cases of tuberculosis of the knee joint. In 36 of these the knee joint was the only joint involved while the remaining had other joints affected. Male patients had the disease three times as frequently as female, there being 33 of the former and 11 of the latter.

The condition is almost invariably treated as rheumatism for months or years before it is

TABLE VII.—END-RESULTS OF TUBERCULOSIS OF THE JOINTS

Rating	Patients	Per cent	Average time after operation
Spines—110 cases			
Excellent	59	54	2 yrs. 6 mos.
Uncertain	19	17	3 yrs. 7 mos.
Died	32	29	*
Knees—44 cases			
Excellent	27	61	2 yrs. 6 mos.
Uncertain	8	19	3 yrs.
Died	9	20	8 mos.
Hips—25 cases			
Excellent			
(a) with solid bony fusion	9		
(b) with 3–5 degrees motion	2		
Uncertain	11	44	4 yrs.
(a) with solid bony fusion	3		
(b) not fused	2		
Died	5	20	2 yrs.
(a) with solid bony fusion	1		
(b) not fused	8		
	9	36	14 mos.
Sacro-iliac joints—16 cases			
Excellent	6	37½	4 yrs. 2 mos.
Uncertain	3	19	2 yrs. 6 mos.
Died	7	43½	†
Tarsal joints—13 cases			
Excellent	9	70	3 yrs. 6 mos.
Uncertain	1	7	6 mos.
Died	3	23	6 mos.
Ankle joints—4 cases			
Excellent	3	75	2 yrs.
Uncertain	1	25	3 yrs.
Upper extremity joints—11 cases			
Excellent—with solid fusion			
Shoulders	2		
Carpus	3		
Elbow	1		
Without solid fusion—elbow	2		
	8	72	3 yrs. 7 mos.
Uncertain—without fusion—elbow	2	18	1 yr.
Died—carpus	1	10	‡
Multiple joint—43 cases			
Excellent	22	51	3 yrs. 6 mos.
Uncertain	7	16.5	3 yrs. 8 mos.
Died	14	32.5	9 mos.
10 patients of the multiple joint series who had a second joint operated upon			
Excellent	7	70	Over 5 yrs.
Uncertain	1	10	3 yrs.
Died	2	20	(a) 13 mos. (b) 3 days—embolus

*10 months (except 3 cardiacs who died within 24 hours, and 1 at 17 days).
†8 months except 1 embolic death at 60 hours after carpal fusion.
‡60 hours after operation, of embolus (this death is also noted under sacro-iliac cases).

recognized. The average duration of symptoms before operation was 4 years. Two patients had had their disease for 19 years and 1 for 14 years.

Age. The age incidence of this group of 44 patients is fairly evenly distributed in the first 5 decades (see Table VI). Average age at onset was 22 years.

Diagnosis. The diagnosis was made by physical examination, roentgenographic study, and a study of excised tissue. The diagnosis was proved positive by tissue examination in 35 of 44 patients. Six cases were explored, found negative for tuberculosis, and not fused.

Sinuses. Sinuses were present in 21 of the 44 patients with knee joint tuberculosis.

Operative and postoperative treatment. The operation employed was a fusion of the knee joint, the patella being used as a peg fitted into a prepared mortice in the femur and tibia as described by Hibbs. After operation, a plaster-of-paris spica was applied for 6 to 8 weeks, followed by a long leg circular plaster until fusion became solid. The length of time required to accomplish bony fusion varied from 4 months to a year or more. The progress of fusion was checked by clinical examination and roentgenogram. Weight bearing with a long circular plaster splint was permitted after 6 months, if the patient's general condition warranted it.

We have had occasion to observe several children whose knee joints had been *resected* at other hospitals and as they reached full growth the leg with the resected knee was between 6 and 7 inches shorter than the sound leg with the disease still active. I should like again to condemn utterly this procedure in children.

End-results. A knee fusion was done in 41 cases and an amputation in 3 cases in which fungating sinuses made any attempt to save the extremity impossible. Fusion became solid in 34 of 35 surviving patients on whom it was attempted, though 4 of them required a second operation. Fusion was also solid in 2 patients who died of their systemic disease 2 or 3 years after operation. This testifies to the efficacy of the operative procedure. The end-results are shown in Table VII. Seven patients who failed to attain fusion of the knee joint were all fatal cases, overwhelmed by their tuberculosis and profuse suppuration. Of 21 patients with sinuses, 18 survived. We have had the discouraging experience of sending a patient home in excellent condition with a solidly fused knee and then watched him die 2½ years later of tuberculosis of the meninges. Mortality was highest in the third decade (see Table IX).

Fusion in children. There were 3 young patients 6, 3, and 2 years of age, respectively. Each had a large fungating synovial type of disease. Fusion was obtained in each of these patients though 2 required a second operation to make solid bony contact. The diseased leg in these youngsters has kept up in growth with the sound leg. Fusion on these small knee joints is technically difficult but worth attempting, when we consider that 4 of our patients with this disease

TABLE VIII.—MORTALITY IN PATIENTS WITH TUBERCULOSIS OF SPINE IN RELATION TO THE AREA OF THE SPINE INVOLVED

	Number of patients	Died	Mortality per cent
Cervical	3	1	33
Cervicodorsal	4	1	25
Dorsal	60	16	26.6
Dorsolumbar	12	3	25
Lumbar	30	10	33
Lumbosacral	5	1	20

entered the hospital as infants and spent 14, 8, 7, and 6 years, respectively, an average of about 9 years each, while various forms of immobilization were tried. At the end of this period each of these patients had active knee joint tuberculosis. In each instance a fusion was done and the patient was discharged from the hospital within a year after operation.

Mortality in relation to systemic tuberculosis is shown in Table V. Cases with positive sputum or metastatic spread to other organs furnished 88 per cent of the total mortality. The one death in a favorable group was from amyloid disease.

TUBERCULOSIS OF THE HIP JOINT

There were 25 patients in this series with tuberculosis of the hip joint, a majority of them males. The right hip was involved 15 times and the left 10 times.

The age of this group was relatively young, 88 per cent of them being under 30 years. The youngest was 3 years and the oldest 41 years (see Table VI). The average age at onset was 13 years.

Duration of the disease. The average duration of the disease prior to operation was about 4 years. Three cases had persisted 10, 15, and 10 years, respectively.

Diagnosis. The diagnosis was made by clinical examination, showing spasm, limited motion, flexion and adduction deformity, limp, etc., and by roentgenographic evidence of a destructive arthritis involving the head of the femur and at times the acetabulum. In most instances the disease had advanced to the stage of bone destruction, but in a few instances it was limited to the synovia. In one instance, a preliminary biopsy was done.

In 18 of the 25 fusions, the tissue removed at operation proved the diagnosis to be tuberculosis. Due to massive destruction, fusion of the unproved joints was completed.

Abscess and sinuses. Abscess was present in 21 of the 25 patients. Sinuses were present in 12 patients prior to operation and developed in 3 others after operation. These facts, I believe, support the contention that the group was an unfavorable one on which to get brilliant surgical results.

Treatment. In our experience fusion of the hip joint in tuberculosis is not easily accomplished. In case there is massive destruction of the head and neck of the femur due to long standing disease, giving an unstable joint, it is doubtful if fusion can be accomplished by any of the methods that have been described.

An operative fusion of the hip was attempted on each of these 25 patients. The Hibbs type—the great trochanter and a piece of the shaft of the femur being rotated on the muscle attachment and driven into the ilium just above the acetabulum—was employed in 10 cases. In the 15 remaining cases a large mass of ilium was used to form a bridge from a deep socket in the femur to the denuded ilium. This type of fusion was first done in 1927. With the Hibbs method, we had only 3 successful fusions in 10 attempts, whereas by the second method there were 10 successful fusions in 15 attempts.

We have immobilized the hip by a double plaster-of-paris spica for 4 to 6 months and then applied a single spica for an additional period until we felt that fusion was solid by clinical and roentgenographic examination or else until we abandoned hope of fusion.

End-results. These are summarized in Table VII.

Subtracting the 8 unfused hips that terminated fatally, the operation was technically successful in 13 of 17 cases, or 76 per cent. Two cases required secondary operations. The failure to obtain a higher percentage of operative success was almost certainly due to the presence of sinuses in 50 per cent of the cases.

Analysis of mortality of 25 patients with hip joint tuberculosis. There were 3 patients who died within 36 hours after the operation of whom 2 were adults with cardiac disease. One patient discharged from the hospital as an excellent result with a solidly fused hip, died of tuberculosis of the meninges 3½ years after operation. Sixty-six per cent of the total mortality occurred in patients with positive sputum or metastatic spread to other organs. The 3 deaths in the more favorable groups were due to cardiac disease twice and to amyloid disease once (see Table V).

Age. The age in relation to mortality shows the third decade leading again (see Table IX).

TUBERCULOSIS OF THE SACRO-ILIAC JOINT

In this series there were 16 patients with tuberculosis of the sacro-iliac joint, with the disease

TABLE IX.—ANALYSIS OF DEATHS IN RELATION TO AGE IN THE 3 LARGER GROUPS

Decade	Tuberculosis of spine			Tuberculosis of knee joint			Tuberculosis of hip joint		
	Number of patients	Died	Mortality per cent	Number of patients	Died	Mortality per cent	Number of patients	Died	Mortality per cent
1–10	24	1	4.16	4	0	0	9	3	33⅓
11–20	20	6	30	12	2	16.6	6	1	16.6
21–30	26	12	46	15	6	40	7	4	57
31–40	17	6	35	3	1	33⅓	2	1	50
41–50	14	4	28	10	0	0	1	0	0
51–60	8	3	37.5	0	0	0	0	0	0
Over 60	1	0	0	0	0	0	0	0	0
Total	110	32		44	9		25	9	

in the right joint in 12 instances and in the left in 5. Seven were males and 9 females. Only 6 of these patients had tuberculosis of the sacro-iliac joint as the sole joint manifestation while 10 of them had other joints involved. Of these, 1 had both sacro-iliac joints invaded.

Age. No patient was under 11 years of age while 75 per cent were between the ages of 11 and 30 years (see Table VI). The average age at onset of disease was 24 years.

Diagnosis. The diagnosis was made by physical examination and roentgenogram. The chief complaint was low backache and stiffness. On examination they showed marked muscle spasm, pain on straight leg raising and extreme rotation of the hip, the pain being referred to the joint involved. The roentgenogram showed a destructive arthritis of the joint with marked atrophy of the adjacent ilium and sacrum, clear cut in some cases, indefinite in others.

The diagnosis was proved positive by excised tissue in 13 of the 16 patients.

Abscess was present in 31 per cent of the patients and sinuses formed in 37 per cent.

Surgical treatment. An operative fusion was performed on each of these 16 patients, extra-articular in 13 and articular in 4 instances. The extra-articular fusion was performed by exposing the ilium and sacrum subperiosteally above and below the posterior superior iliac spine and then denuding the non-articular area between the ilium and the sacrum. A block of bone from the iliac crest was wedged into a prepared bed between the ilium and the sacrum and bone chips were packed around the entire denuded area. This method was used from 1926 to 1930. It is not as satisfactory, I believe, as the articular type of operation of Smith-Peterson which is used at present.

End-results. These are summarized in Table VII.

The presence of fusion in the sacro-iliac joint is difficult to determine with certainty, in many instances, either clinically or by roentgenographic examination. However, we believe that all our surviving patients have solid fusion.

Analysis of the mortality of 16 patients with sacro-iliac joint tuberculosis. Our experience in this relatively small series has made us feel that sacro-iliac joint tuberculosis is a very serious manifestation with a mortality which is high for joint tuberculosis.

In this series, 75 per cent of the patients with tuberculosis of the sacro-iliac joint were in the groups with active pulmonary disease, positive sputum, cavities in the lungs, etc., or if they had a negative sputum, their disease manifested itself by metastatic spread to other organs. These patients furnished 100 per cent of the mortality (see Table V).

TUBERCULOSIS OF THE TARSAL JOINTS

There were 13 cases of tarsal joint tuberculosis in this series, in one of which the ankle joint was also involved. The diseased joints were distributed as follows: 5 subastragaloid joints; 4 tarsometatarsal joints; 3 entire tarsus; 1 calcaneus.

The disease was distributed almost equally between the sexes, with 7 males and 6 females. The right foot was involved 4 times, and the left 9 times.

Age. The distribution by age showed a wide spread from the first to the sixth decade (see Table VI). The average age at onset was 26 years.

Duration of the disease was long, the average being 3 years and 9 months before operation.

Diagnosis. The diagnosis was made by physical examination; swelling, tenderness, marked muscle spasm. The roentgenogram showed a destructive arthritis with extensive atrophy. Sinuses were

TABLE X.—CASES WITH MULTIPLE JOINT FOCI WITH SUMMARY OF THE JOINTS INVOLVED

Joint or focus	Operation performed at Sea View	Elsewhere	No operation
Spine	25	1	3
Hip	5	..	5
Knee	8	..	2
Sacro-iliac	9	..	..
Both sacro-iliac	1	..	..
Second spine focus	3	..	2
Shoulder	1	..	2*
Elbow	2	1	7
Wrist	3	..	3
Tarsus	2	..	4
Ankle	..	..	1
Dactylitis	2	..	3
Tendon sheath	2	..	..
Ribs	1	..	3
Scapula	..	..	1
Maxilla	..	..	1
Total	64	2	37

*Shoulders in one patient.

present in 9 out of 13 patients. The diagnosis was proved by examination of the tissue removed in 7 of the 13 cases.

One patient in addition to tuberculosis of the tarsus had most extensive gout. His entire forefoot showed masses of uric acid crystals all through the metatarsal bones. This patient died of advanced pulmonary tuberculosis.

Surgical treatment. Amputation through the upper third of the leg was resorted to in 4 cases with extensive widespread disease with abscess and sinuses. These patients all convalesced uneventfully. Fusion of tarsal joints was done by simple removal of the articular cartilage. Postoperative fixation by plaster boot was maintained for 4 to 6 months.

End-results. These are summarized in Tables V and VII.

Fusion of the involved joints was performed in 9 cases and as a procedure it was 100 per cent successful, proved by physical examination and roentgenogram, but it failed to alter the balance in favor of 3 patients who died of generalized tuberculosis. One uncertain case left the hospital 6 months after operation with solidly fused tarsus but active pulmonary tuberculosis, and has not been heard of since.

Analysis of mortality. The entire mortality was in the group with positive sputum.

TUBERCULOSIS OF THE ANKLE JOINT

There were but 4 cases of tuberculosis of the ankle joint in this series. In 3 of these patients the ankle was the only joint involved while in one of them the subastragaloid joint was also infected.

TABLE XI.—MORTALITY IN THE VARIOUS AGE GROUPS IN 210 PATIENTS WITH VARIOUS TYPES OF JOINT TUBERCULOSIS

	Total	Died	Mortality per cent
1–10	48	7	14.8
11–20	46	10	21.7
21–30	52	24	46
31–40	28	10	35.7
41–50	26	5	15
51–60	9	3	33⅓
Over 60	1	0	0
Total	210	59	28.5

These 4 were equally divided between right and left feet but the males outnumbered the females 3 to 1.

Age. Three of the patients were in the second decade and the remaining one was 58 years of age. The average age at onset was 25 years.

Duration of the disease. The duration of the disease was less than 1 year in 3 patients and the other patient had had the disease for 2 years.

Diagnosis. The diagnosis was made by physical examination and roentgenogram and was proved correct by the excised tissue in all 4 instances.

Surgical treatment. Amputation was resorted to in 1 patient and fusion in 3 patients. The operative procedure employed in fusing the ankle joint was as follows: The articular cartilage of the entire ankle joint, including the medial and lateral malleoli, was removed. Into the raw joint surfaces was packed a mass of bone chips from the tibia and the astragalus. A bone graft from the adjacent tibia was placed across the front of the joint from a groove in the tibia to a similarly prepared groove in the astragalus. A plaster-of-paris boot was applied with the foot at an angle of 100

TABLE XII.—COMBINED END-RESULTS FOR 224 JOINTS INVOLVED

	Excellent		Uncertain		Died		Total
	Number	Per cent	Number	Per cent	Number	Per cent	
Spines	59	54	19	17	32	29	110
Knees	27	61	8	19	9	20	44
Hips	11	44	5	20	9	36	25
Sacro-iliac	6	37.5	3	19	7	43.5	16
Tarsus	9	70	1	7	3	23	13
Ankle	3	75	1	25	0	0	4
Symphysis pubis	1	100	0	0	0	0	1
Upper extremity	8	72	2	18	1	10	11
Total	124	55.3	39	17.7	61	27	224

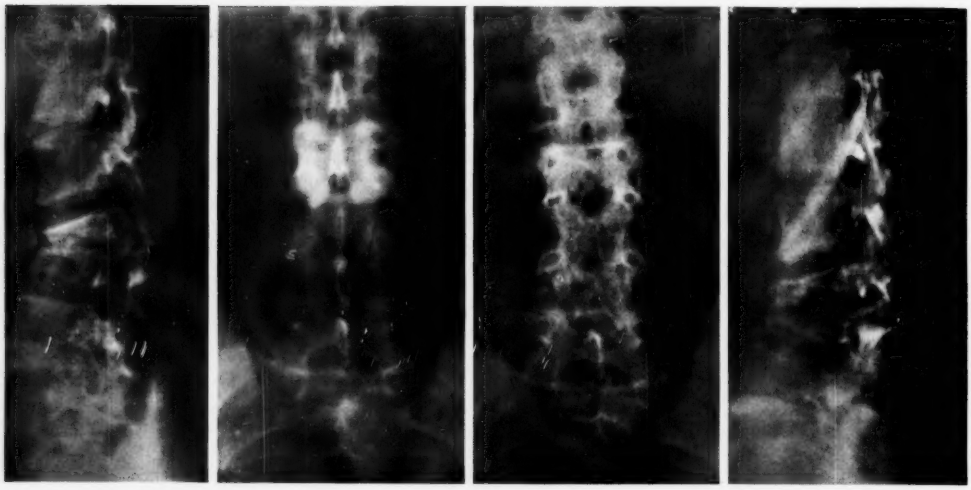

Fig. 1. Case 1.

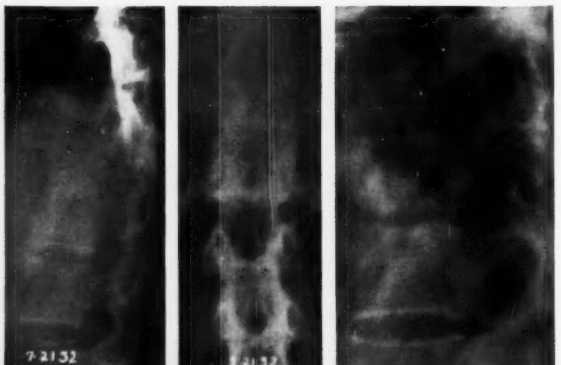

Fig. 2. Case 2.

degrees to the long axis of the leg. This immobilization was maintained for from 4 to 6 months.

End-results. The fusion was completely successful in all 3 cases. Three patients rated as excellent had had no evidence of pulmonary tuberculosis. One rated as uncertain has a 100 per cent result as far as ankle fusion is concerned but he has an active pulmonary tuberculosis and is at present in a sanatorium under treatment. He may well achieve an excellent result in time (see Table VII).

TUBERCULOSIS OF THE SYMPHYSIS PUBIS

This joint was involved in 1 case in this series, a Greek woman in her early thirties. There was a persisting sinus which had not healed for 3 years. Her general condition was excellent so it was decided to attempt surgical intervention. The diseased bone surface was excised and a bone block from the ilium was inserted into a prepared slot between the 2 pubic bones; a number of chips were added. The block and all the chips were extruded as sequestra and the condition cleared up with healing of the sinus and has remained healed and symptomless for 3 years after operation.

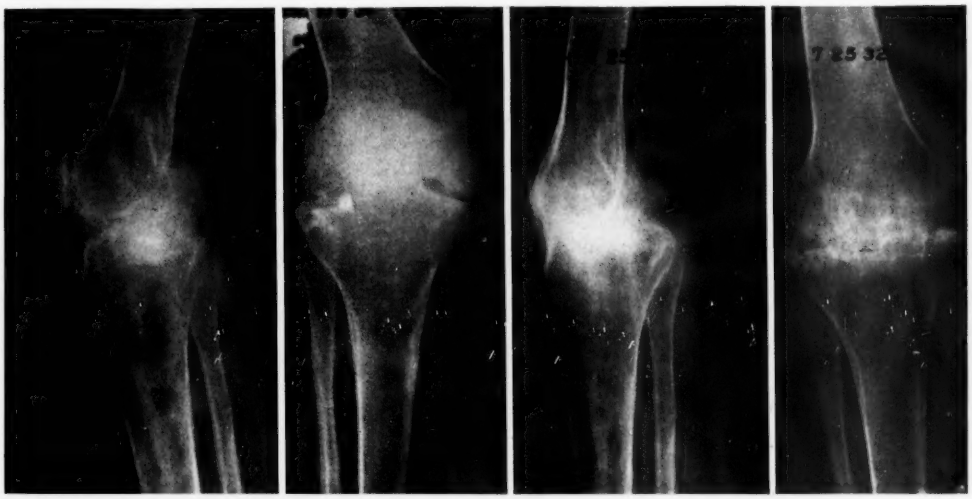

Fig. 3. Case 3.

A simple resection of the diseased soft tissue and the bone would have been infinitely better in this case than the attempted fusion and if there were any subsequent instability, a fusion of the two sacro-iliac joints could readily be done.

This patient had no evidence of pulmonary disease. We have seen tuberculosis of this joint in several other instances but inoperable because of extensive pulmonary disease or widespread dissemination.

UPPER EXTREMITY JOINT TUBERCULOSIS

That less than 8 per cent of the total joints involved with tuberculosis were in the upper extremity must be explained on the basis of traumata incident to weight bearing.

In this series there were 17 patients with upper extremity joint tuberculosis, one having both shoulders affected. In only 5 of these was the upper extremity joint the only one involved. The 12 remaining had various other joints involved. Ten were elbows, 4 shoulders, and 4 carpal or wrist joints.

Of these 18 joints, only 11 were treated surgically. Those not treated surgically at Sea View Hospital included 4 elbows which were terminal manifestations, 1 elbow which had been previously successfully resected, and 1 bilateral shoulder case which also had a hip and spine involved. The shoulder symptoms subsided to a point where they were not considered important enough to warrant operative treatment.

Age. The highest age incidence was in the second and third decade (see Table VI). The average age at onset was 22 years.

Duration of disease. The average duration of the disease was 3 years. One patient had an active elbow joint for 15 years. Only 5 of the patients had complained of their symptoms for 6 months or less.

The operative cases were as follows:

1. *Shoulder joint.* There were 2 cases, each proved positive by excised tissue. Both had draining sinuses. Both were treated by operative fusion which was successful. The end-result was excellent in both 3 and 7 years after operation, respectively.

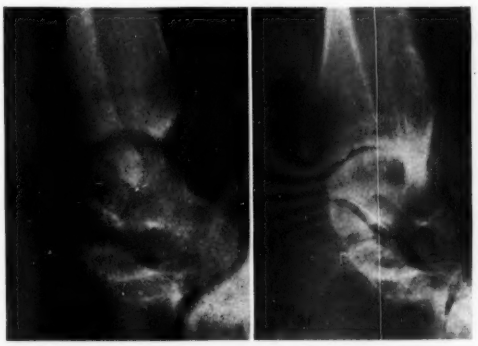

Fig. 4. Case 4.

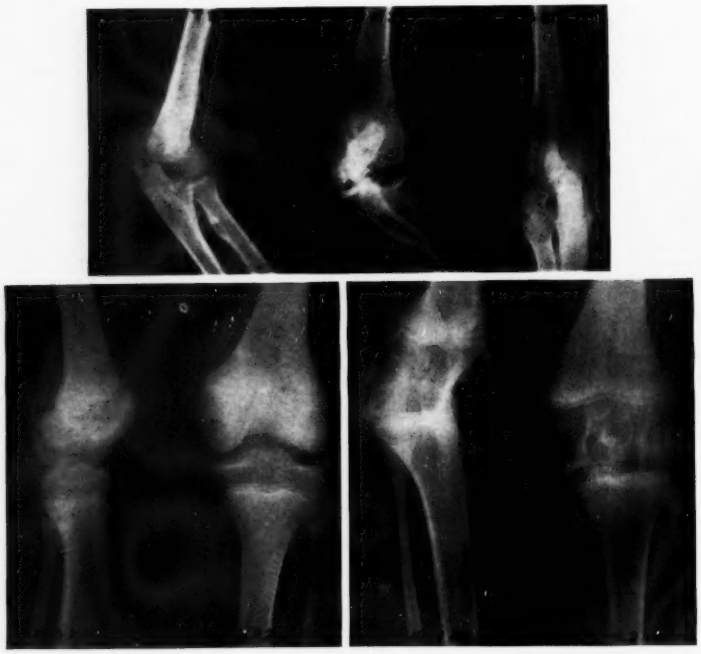

Fig. 5. Case 5.

2. *Elbow joint.* There were 5 cases of elbow joint tuberculosis in this group with a diagnosis proved by tissue examination on 4 of the 5. Sinuses were present in the majority. Fusion of the elbow joint was attempted on all 5. One became solid, 2 developed pseudo-arthrosis with quiescent disease, 1 came to amputation and 1, a fungating tuberculous elbow, is dying of amyloidosis.

Fig. 6. Case 6.

Technically, fusion of the elbow joint is difficult in favorable cases and should not be attempted in unfavorable ones. I now believe a careful resection may be a better and more logical procedure in this non weight bearing joint. Our record of only 1 successful fusion in 5 attempts leaves much to be desired.

3. *Wrist and carpal joints.* There were 4 cases of wrist or carpal joint tuberculosis, all proved positive by excised tissue.

Fusion was performed in 3 with excellent results and solid bony fusion, though one required 2 subsequent operations because of prolonged suppuration. The one failure patient died of an embolus 3 days after operation.

End-results. These are summarized in Tables V and VII.

The mortality in this group is low but the 2 uncertain cases will probably raise it.

CASES WITH MULTIPLE JOINT FOCI

There were 43 patients who presented multiple tuberculous joint, tendon sheath, and bone, foci. Among these 43 patients were 113 separate foci, 64 of which were operated upon at Sea View Hospital, 2 elsewhere, and 37 foci were inoperable,

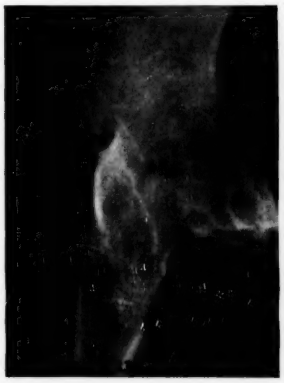

Fig. 7. Case 7.

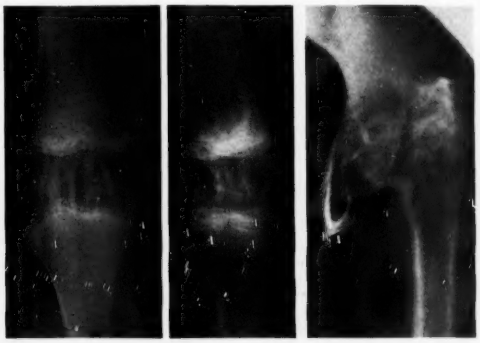

Fig. 8. Case 8.

being either terminal or quiescent manifestations (see Table X).

Eighty per cent of the patients were under 30 years of age (see Table VI). The average duration of disease was over 4 years, though 2 cases had persisted for 33 years or more, 4 others over 10 years. Only 6 had symptoms for 6 months or less.

Twenty-five of the patients had spinal involvement. Nine patients had sacro-iliac involvement, which as has been shown, carries with it a very bad prognosis. Of 14 fatal cases in this group, only 2 were worthy of attempt at salvage by operation on a second focus.

End-results. These are summarized in Tables V and VII.

Even with this massively involved group of cases, when the derelicts are excluded, such as one patient who had a second spine focus, dactylitis, tendon sheath and rib involvement, the results were surprisingly good in those patients chosen for operation.

Of the 10 patients in this group who had two or more joints operated upon, 70 per cent had excellent results over 5 years after operation (Table VII).

The 7 excellent results include some of our most brilliant and satisfactory cases. They are as follows: (1) a spine and sacro-iliac joint; (2) a right knee and left hip joint; (3) a carpus, tarsus and spine; (4) a spine and hip joint; (5) a shoulder, sacro-iliac and spine (2 foci); (6) a knee and elbow joint; (7) a knee and carpus. All of these joints are solidly fused and the patients are well and out of the hospital.

One of these patients had pulmonary tuberculosis with positive sputum; 4 had pulmonary tuberculosis with negative sputum but had metastatic spread to other organs. One had negative sputum with no metastatic spread. Only 1 patient of the 7 had no evidence of pulmonary disease.

These excellent results make us reluctant to condemn every patient with positive sputum or metastatic spread. They are brands snatched from the burning.

SUMMARY

Involvement of the joints is a serious manifestation of tuberculosis. It is essentially a disease of early adult life. The average age of onset for these 210 patients was 22 years. The mortality among these patients was 28.5 per cent and will undoubtedly be higher as time goes on and the number of deaths is augmented from the list of those we consider as uncertain. In the first decade of life the mortality in this group was only 14.8 per cent but in the third decade the disease took an enormous toll of our patients, as the mortality mounted to 46 per cent (see Table XI).

From the surgeon's standpoint the treatment of this disease is beset by many disappointments which are offset by the restoration to a relatively normal life of a majority of the patients. The operative treatment in joint tuberculosis aims to eliminate, by amputation, a dangerous focus which cannot be saved, or to attempt to heal by putting the involved joint at complete rest by operative fusion. In this latter case the surgeon merely creates a more favorable situation for healing and the patient recovers if the balance is thereby turned in his favor. The statement of Ambroise Paré, "I dressed him, God healed him" (*Je le pansay, Dieu le guarit*)[1] never held more truth than in the treatment of this disease.

[1] Quoted from Garrison's *History of Medicine*, 4th ed. p. 225.

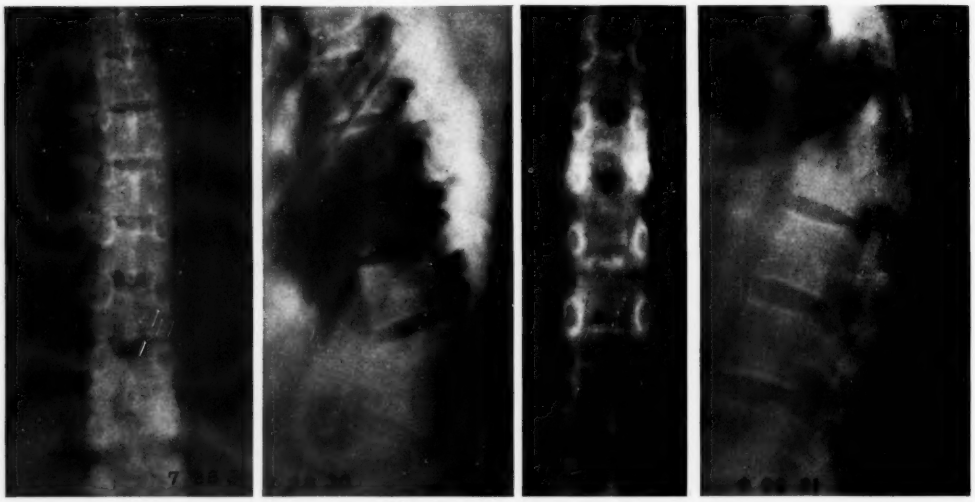

Fig. 9. Case 9.

The fate of the patient depends upon the nature and extent of his tuberculosis. Those patients with positive sputum, caseous pneumonic lesions, etc., and those with pulmonary disease, negative sputum, but with metastatic spread to other organs have shown a mortality of 52 per cent. On the other hand, those patients with inactive pulmonary disease and no metastatic spread to other organs and those with no evidence of pulmonary disease, have shown a mortality that is only 9 per cent.

Our mortality rate of 47 per cent for patients with active pulmonary disease, caseous pneumonic tuberculosis (see Table V, Group A) should be compared with the mortality in similar cases uncomplicated with joint manifestations.

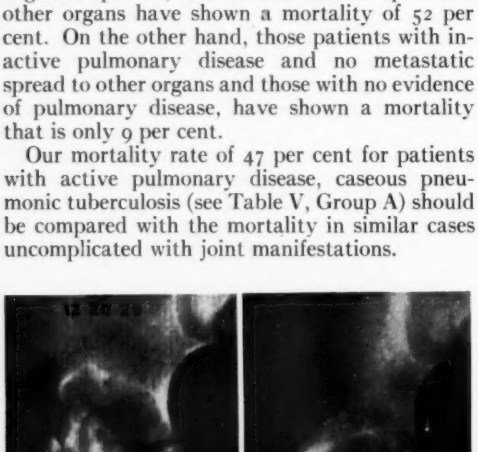

Fig. 10. Case 10.

David Ulmar reports, in a personal communication, that, at Metropolitan Hospital, New York City, of 700 consecutive admissions with caseous pneumonic tuberculosis, 68 per cent died within a year and at the end of a 10 year period less than 1 per cent survived.

From the Rhode Island State Sanitarium, Barnes and Barnes have reported a death rate for patients with tuberculosis showing cavities in the lungs of 80 per cent within 1 year of admission.

A consideration of these facts amply explains the high mortality in those patients in this series with active pulmonary disease.

Another factor which adversely influences the end-result is prolonged suppuration from sinuses with resulting amyloid disease.

The only joints in our experience which tend to fuse spontaneously are those of the spine. This occurs only in 22 per cent of the cases and then only after many years and tremendous deformity have ensued.

A study of the combined end-results of the 224 joints treated shows the following facts (see Table XII): The upper extremity joints, tarsal and ankle joints had the highest percentage of excellent results, each group 70 per cent or better. The knee joint cases showed 61 per cent and the spine cases 54 per cent excellent results. The percentage of excellent results fell to 44 per cent in the hip joint cases and to 37.5 per cent in the sacro-iliac joint cases. As the excellent results declined, the mortality rose. The sacro-iliac joint with a mor-

tality of 43.5 per cent is, in our experience, the gravest form of joint tuberculosis while the knee joint and upper extremity joints offer the best prognosis.

The work in this series of patients is almost entirely salvage. They represent a selected group, selected from the standpoint of probable unfavorable outcome. Coming from the lower classes of society, from poverty and disease, of less fit racial strains, they have been discarded by most of the city's hospitals and sent to Sea View Hospital for ultimate disposal. This is joint tuberculosis such as is not usually reported. The 300 odd cases which we rejected as unsuitable for surgery, presented the appalling mortality of over 80 per cent.

Our results in these 224 operative joints showed 124, or 55.3 per cent, which can be honestly rated as excellent, 39, or 17.7 per cent, uncertain, and 61, or 27.2 per cent, died. The fact that only a little over half can be rated excellent should not be considered remarkable but what should be stressed rather, is that so many patients can be salvaged from such an unfavorable group.

CASE REPORTS

The following 10 case reports, in brief abstract, with roentgenograms are submitted as typical of varying phases of the problems encountered in these 210 patients.

CASE 1. C. F. A young woman of 23 years with a history of backache of over 1 year's duration (Fig. 1).

The pre-operative roentgenograms show definite destruction of the second and third lumbar vertebræ. A spine fusion was done extending from the twelfth dorsal to the fifth lumbar, inclusive. Postoperative course was quite uneventful. The spine was solidly fused clinically 6 months after operation and the roentgenograms taken 10 months after operation prove this solid fusion. This patient had no evidence of pulmonary involvement and was discharged as an excellent result 10 months after operation.

CASE 2. M. C., 61–28. A young woman of 25 years who came to Sea View Hospital in 1926 (Fig. 2). She gave a history of pleurisy 6 months before admission and shortly after this she developed a painful swelling of the right shoulder.

A diagnosis of tuberculosis of the shoulder joint was made and at time of operative fusion in November, 1926, tissue removed confirmed the diagnosis. She had an occasional positive sputum. In November, 1927, she developed a lesion in the spine, involving the twelfth dorsal and first lumbar. This area was operatively fused in February, 1928. The left sacro-iliac joint was next invaded and operatively fused. The patient left the hospital and has been home well for 4½ years. When she last appeared for follow-up examination in February, 1933, a small kyphos was found above her former spine fusion and she complained of a little mid-dorsal backache. The roentgenogram showed a lesion involving ninth and tenth dorsal vertebræ.

These roentgenograms taken in 1933 show a fused shoulder joint, a fused dorsolumbar spine, and a fused left sacro-iliac joint and the latest focus involving the ninth and tenth dorsal verteberæ which has subsequently been fused. Attention is directed to the solidity of the lumbar spine fusion 5 years after operation. The pre-operative roentgenograms were destroyed along with other inflammable films several years ago.

This is shown as an example of diffuse spread, with multiple joint lesions in a patient whose resistance is very high. Her general condition is excellent.

CASE 3. G. H., 5122–31. A man of 37 years who since the age of 4 years has had tuberculosis of the spine with tremendous deformity resulting. He came to Sea View Hospital in 1931 with a 10 year history of painful swelling in the right knee. This was diagnosed and at operation proved to be tuberculosis. A knee fusion was done in November, 1931, and also the tendon sheath of the right middle finger was excised as he had a proved tuberculosis of this sheath.

This patient was discharged from the hospital 6 months after operation. He wore a circular plaster splint for 3 months longer. He has a solidly fused knee joint and is in excellent condition 2½ years after operation.

The roentgenograms show the right knee before operation in October, 1931, and after operation in July, 1932, (Fig. 3).

CASE 4. W. R., 3166–30. This patient, a boy of 17 years with active pulmonary disease and positive sputum, came to Sea View Hospital in 1929. He subsequently developed a painful swelling of the left ankle joint. This was diagnosed tuberculosis. A fusion of the ankle joint was performed in May, 1930, and the tissue removed confirmed the diagnosis. The fusion was solid and patient was walking without support 9 months after operation.

His pulmonary disease has been treated with pneumothorax and sputum has been negative. He is still under treatment for pulmonary tuberculosis at Otisville Sanitarium, with a very good prognosis.

The roentgenograms show the ankle joint in April, 1930, before operation and in January, 1931, after operation (Fig. 4).

CASE 5. H. F., 20–30. A colored boy of 11 years who came to Sea View Hospital in 1928 and who developed pain and swelling in the left knee and a little later in the right elbow. These complaints were noted subsequent to his admission to hospital for a non-productive cough and loss of weight. He also had a keratitis. His general condition improved steadily. His sputum was scanty and always negative.

In March, 1929, the knee was operatively fused and the elbow was similarly treated in July, 1929. Tissue removed at both of these operations confirmed the pre-operative diagnosis of tuberculosis. Both operations were successful. The knee joint, however, bent through the epiphyseal line into recurvatum and had to be straightened with an osteotomy of the femur. The patient is in excellent condition at home with both joints solidly fused, 5 years after operation.

The roentgenograms show the elbow joint in July, 1928, before operation and in September, 1931, after operation, and the knee joint in June, 1928, before operation and in October, 1930, after operation (Fig. 5). Attention is directed to the solid bony fusion of the knee joint with the recurvatum deformity which occurred through ununited epiphyses.

CASE 6. C. M., 4515–31. A colored girl of 12 years who was admitted to Sea View Hospital in 1931, complaining of

pain and stiffness in the left hip for 18 months prior to admission. A diagnosis of tuberculosis of the hip was made and subsequently confirmed by tissue examined. A fusion of the hip joint was done in May, 1931. A large mass of iliac bone was used as a graft from the femur to the ilium. The patient wore a double plaster-of-paris spica bandage for 4 months and then a single spica. She began to bear weight 5 months after operation and at 8 months all support was removed and fusion was definitely solid.

The patient had no evidence of pulmonary tuberculosis. She has been home over a year and continues well, an excellent result 2 years after operation.

The roentgenograms show the left hip before operation in May, 1931, and after operation in May, 1932. Excellent bony fusion (Fig. 6).

CASE 7. M. B., 177-28. An Italian girl of 16 who was admitted to Sea View Hospital in 1927 with extensive tuberculosis of the spine and of the right hip joint. The spine was explored and spontaneous fusion was found from seventh dorsal to second lumbar. Four vertebræ above and 1 below were added because of the severity of the kyphos and extent of the disease. In August, 1927, a hip fusion was done, an iliac graft being used to bridge the gap from the femur to the ilium. Tissue removed confirmed the diagnosis of tuberculosis. A plaster spica was worn for 6 months and, during the last month of this period, the patient began to bear weight. She left the hospital 9 months after operation. When seen in follow-up in 1933, 6 years after her hip fusion, her general condition was excellent; hip was solidly fused.

The roentgenogram taken in 1933 shows a solid fusion of the right hip (Fig. 7). Her pre-operative pictures were destroyed because they were inflammable film.

CASE 8. A. C., 95-28. A boy of 7 years, admitted to Sea View Hospital in September, 1925. He had been in various hospitals for 5½ years because of pain and stiffness in his left hip and later his right knee. There was active disease in both these joints. In March, 1926, a fusion of the right knee was performed and in April, 1926, a fusion of the left hip was performed. Tissue removed at each of these operations showed tuberculosis. He wore a circular plaster on the right knee for 6 months when fusion was found to be solid and the hip joint was supported by a plaster spica for 8 months, at which time the joint was definitely fused. He was discharged from the hospital 22 months after operation in excellent condition, and has remained well, an excellent result, 7 years after operation.

The roentgenograms show the end-result of the hip and knee in March, 1933 (Fig. 8). Pre-operative films were destroyed. The patient is shown as an excellent result in a multiple foci case, the patient previously having had 5½ years of conservative treatment, at the end of which period he was bedridden with active disease in both hip and knee joint. This boy has grown tremendously since operation and the involved joints that were fused have kept pace with the uninvolved joints.

CASE 9. E. G., 962-28. A woman of 24 years, admitted to Sea View Hospital first in 1926 and again in 1928 for pulmonary tuberculosis. She had an active pulmonary lesion with positive sputum until March, 1929. From that date until her discharge from hospital in October, 1931, her sputum was negative. She developed lower dorsal backache and a slight kyphos while under treatment for pulmonary tuberculosis. In September, 1930, definite disease was noted by X-ray in eleventh and twelfth dorsal vertebræ and a spine fusion was done extending from ninth dorsal to second lumbar.

Postoperative course was uneventful. The patient left the hospital 13 months after operation with solidly fused spine and her pulmonary condition arrested.

The roentgenograms show the lesion in eleventh and twelfth dorsal before operation, July, 1930, and 1 year after operation in September, 1931. Bony fusion can readily be seen and fusion of the bodies is taking place (Fig. 9).

CASE 10. N. L., 1133-27. This patient, a female infant, was admitted to Sea View Hospital at the age of 2 months because her mother was a patient with pulmonary tuberculosis. The mother died of her disease. This little patient had active pulmonary disease and a cold abscess of the left thigh. Her earliest roentgenograms showed erosion of the femur and tissue from the abscess cavity showed tuberculosis. The left hip joint became involved by direct extension from the femoral shaft.

A fusion of the hip joint was performed in January, 1930, when patient was 3 years old. This fusion was clinically solid 10 months after operation. For the past 2 years she has had no symptoms. She is in excellent condition and her fused hip allows her to run around at will.

The roentgenograms show marked cavitation of the femoral shaft and acetabulum before operation in the preoperative view of December, 1929, and the postoperative view of August, 1932, shows solid fusion across the epiphysis with recalcification of the femur and ilium. An excellent result in a seemingly hopeless case (Fig. 10).

MEDICAL TREATMENT OF AMEBIC INFECTIONS OF THE LIVER

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THE hepatic complications of intestinal amebiasis include acute hepatitis and abscess. Acute hepatitis results in the occurrence of chills, sweats, fever, leucocytosis, and swelling and tenderness of the liver. These symptoms constitute the earliest response to invasion of the liver by Endameba histolytica, but, being nonspecific in character, they rarely lead to a correct diagnosis even during an epidemic of amebiasis. When correctly diagnosed the treatment is everywhere regarded as being a purely medical problem. Delay in instituting medical treatment permits an increase in the size of multiple areas of softening which may fuse or coalesce to form one or more abscesses. The symptoms of the stage of abscess do not differ materially from those of the stage of hepatitis. The physical signs, however, may show immobilization or deformity of the diaphragm, displacement of the liver, or the presence of a mass in the upper abdomen or right lumbar region. The treatment for this stage of amebic infection, unlike that of hepatitis, is almost everywhere surgical. Craig says, "The treatment of amebic abscess of the liver is largely a surgical problem, but as already mentioned the early recognition and treatment of amebic hepatitis and beginning abscess formation is essentially a medical problem." In a recent article, Freund expresses the belief that immediate operation is important in the treatment of amebic abscess of the liver.

That a pyogenic abscess is best treated by surgical measures is not subject to dispute, but a pyogenic abscess and an amebic abscess are not comparable conditions. An amebic abscess is the result of the lytic action of ferments liberated by amebas. The material in an amebic abscess is composed of liquefied liver tissue and red blood cells in various stages of disintegration. Cells of leucocytic origin are not found in an uncomplicated amebic abscess, nor are living amebas present in the material first evacuated, although they may be present in great numbers in the tissue which forms the wall of the cavity. It is by reason of their location in the wall of the cavity that they are enabled to continue their lytic action, thereby constantly enlarging the size of the abscess. An amebic abscess differs from a pyogenic abscess further in that it is caused by a parasite that is easily killed by any one of several drugs introduced into the body at a point remote from the actual site of infection. In view of these facts, surgical evacuation of an amebic abscess of the liver would appear to be an illogical procedure.

Surgical drainage of amebic abscesses of the liver is attended with an extremely high mortality rate, particularly when an open operation is performed. The danger of open operation resides in the fact that secondary bacterial infection is an almost invariable sequel. In 75 of 87 cases of liver abscess reported by Rogers, cultures for pyogenic bacteria made at the time of operation were sterile, although subsequently bacterial infection occurred. Aspiration is less likely to result in secondary bacterial infection since it does not leave an open sinus, but this advantage is almost counterbalanced by its own peculiar shortcomings. Aspiration is an attempt to introduce the point of a needle into a cavity of unknown size, at an unknown depth in the largest organ in the body. Should the needle enter the abscess at the first attempt only a fraction of the sterile liquid contents could be aspirated, and even if the abscess could be evacuated completely, the parasite which caused it has not been disturbed, since it is situated at varying depths in solid liver tissue. Failure to strike the abscess at the first attempt is likely to cause dissemination of the infection throughout the liver as a result of implantation by multiple punctures. I have seen such induced abscesses on several occasions at autopsy. With both open operation and aspiration too much reliance is placed on the assumption that amebic abscesses always occur singly. Numerous instances of multiple abscesses have been reported. Wherein lies the advantage of partially evacuating an amebic abscess, or even evacuating it completely, if another abscess exists that has not been drained? Gesner has collected and reported 56 cases of amebic liver abscess. In 23 cases the abscess was incised and packed with iodoform gauze; in 17 cases the abscess was incised and drained with tubes. In each group there were 5 deaths. Cort reports 530 cases of amebiasis in 97 of which there was hepatic involvement. In 14 cases the abscess was evacuated by aspiration, in 2 by rupture through the lung, and in 1 by rupture into the pleural cavity. The remaining cases were treated medically. He says, "In all cases the injection of emetine brought about amelioration of symptoms and reduction in the size of the liver within 2 to 4 days. In

some cases this reduction was almost incredible." There was only 1 death in the entire series and this case had cardiorenal findings. Cort favors operation only when there is a secondary infection. Murray and Koh report 23 cases of amebic liver abscess, in 3 of which treatment was by emetine alone. Biggam and Ghalioungui report 25 cases treated medically for from 5 to 7 days as a preliminary to aspiration. In 2 of their cases recovery was so prompt that aspiration was not necessary. Manson-Bahr reports 45 cases of amebic liver abscess, in 13 of which cure followed medical treatment with emetine and emetine bismuth iodide.

As a result of the epidemic of amebiasis which occurred in Chicago I had the opportunity of treating 2 cases of amebic liver abscess. Convinced of the dangers of surgery in this condition and convinced that surgical treatment of an uncomplicated amebic abscess is illogical, both cases were treated medically.

CASE 1. F. W., a white male, aged 50 years, referred by Dr. H. E. E. Barnard, was admitted to Wesley Memorial Hospital November 22, 1933. His illness of 6 weeks' duration was initiated by signs of an upper respiratory tract infection followed in 3 weeks by pain and tenderness in the lower right chest and under the right costal margin. The pain was associated with chills and fever. The sequence of events and the physical findings suggested the possibility of pleural effusion and a diagnostic thoracocentesis had been done with negative results. On admission examination revealed a prominence of the right lumbar region with marked tenderness on palpation and percussion. A spherical mass extending downward and forward from beneath the liver could be felt. The lateral margin of the liver was an inch below the costal margin. The chest showed a partial right pneumothorax. The white cell count varied between 21,300 and 38,950 and the temperature between normal and 104 degrees for the first week. The urine was normal. The stools did not contain either trophozoite or encysted forms of Endameba histolytica until after the administration of a saline cathartic. X-ray examination confirmed the presence of a partial right pneumothorax and showed fixation of the right diaphragm. Treatment with emetine hydrochloride was instituted on November 25. The last febrile reaction of 99.2 degrees occurred November 29. On the day of discharge, December 7, neither the mass nor the liver could be felt and all pain and tenderness had disappeared. The past history of the patient indicated the possibility of amebiasis of 12 years' duration with a latent period of 3 years immediately preceding development of the liver abscess.

CASE 2. J. J. S., a white male, aged 39 years, referred by Dr. Ira Greenberg, was examined on March 23, 1934. The illness for which he sought relief began acutely on January 15, 1934, with fever, chills, severe pain in the right side of the abdomen. There followed loss of weight, weakness, and an increase in the pain which had become unbearable. Examination showed emaciation, pallor, a subicteric tint to the scleræ, and prominence of the right upper quadrant of the abdomen. The liver edge was 10 centimeters below the costal margin and was distinctly tender. The spleen could just be felt. White blood cells 13,200; red blood cells, 4,000,000; hemoglobin 58 per cent. The urine was normal. A blood culture remained sterile. Wassermann and Kahn tests were negative. Examination of the stools was negative until after the administration of a saline cathartic. Motile amebas and the Trichomonas hominis were then found. Treatment with emetine was instituted. Recovery was gradual with a disappearance of all symptoms and signs after April 23, the last day on which there was any elevation of temperature.

CONCLUSIONS

Uncomplicated amebic infection of the liver, whether in the stage of acute hepatitis or of abscess, can be treated successfully by medical measures alone. Surgical treatment should be reserved for cases complicated by secondary bacterial infections and for other complications, such as rupture of the abscess into the pleural or peritoneal cavities.

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PINEAL TERATOMAS

With Report of a Case of Operative Removal[1]

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PINEAL tumors are considered to be of extreme rarity, and to constitute but 0.7 per cent of all intracranial tumors (5), which latter in turn are the cause of only 0.06–0.15 per cent of general mortality (20). Even within the assembly of tumors of the pineal gland, however, teratomas are rare; only 25 have been reported in the literature of the past half century; the case presented herein, a teratoma, is possibly unique in its range of histologic structure. Tumors of the pineal region are not only difficult to diagnose (only about 30 per cent of those reported in former years were diagnosed clinically), but even when antemortem diagnosis has been possible, therapy has been considered hopeless. It is true that misdirected cerebellar operations have at times been undertaken (5), but surgery's attitude toward purposeful operative attack upon these tumors has been (Cushing, 1926, 4) that "it is improbable that we shall ever be able successfully to extirpate these lesions, in view of their inaccessible position . . . from all points of view they are most unfavorable." Such a therapeutic impasse is always a challenge to endeavor, however, and in the past decade and a half surgical resource and daring has on 6 occasions successfully accomplished the removal of such tumors.

REPORT OF CASE

D. F., a boy aged 6 years, was referred for examination September 20, 1932, by Dr. Thompson Coberth, The Dalles, Oregon, with complaints of headaches, vomiting, and failing vision.

His birth had been at term, with normal labor, but it had required 35 minutes to induce spontaneous breathing. He was jaundiced for 8 or 10 days in the first month of life; pus was expressed from his tonsils when he was 2 weeks old. He had "influenza" at 4 months and at 6 months; and a bilateral pneumonia when 9 months of age; a cervical lymph gland on the left side was drained when he was 1 year old. He had had almost continuous coryza each winter; he had pertussis when 2 years of age, measles when 4 and again when 6 years of age; with the last attack a myocarditis had been diagnosed and his parents had spent several weeks in the mountains with him, preliminary to a contemplated removal of tonsils almost occluding his pharynx. He had been subject to colic-like attacks during the first several months of life; he had had considerable trouble with his teeth; his appetite was fickle, and he was chronically constipated, requiring laxatives several times a week. Cardio-respiratory and genito-urinary history was negative. He was precociously bright at home; had always been restless in his sleep. His gait was normal, and he "ran like a deer." His best weight had been 40 pounds, 6 pounds under the ideal, but he had recently lost considerably in weight.

For the 3 weeks preceding his present illness he had often had sudden bouts of severe intertemporal and frontal headache, with sharp prostrating pains, occurring most often at evening and night; these had been ameliorated by castor oil catharsis. He had become "nervous" (unusually quick) in his movements; his voice had lost its intonation, he had begun to stutter; "after a few minutes' conversation he would be screaming almost at the top of his voice." The right side of his face did not function as well as the left. A week before examination, after being given one aspirin tablet for headache, his vomiting at frequent intervals had begun; emesis relieved his headaches. For 5 days he had had attacks of severe epigastric cramps, bad enough to make him "rave with pain"; these spells were apparently independent of the headaches. Three days before examination, his left eye had turned inward, and he had complained of double vision at such times as he could see; light hurt his eyes, and his vision had become foggy, with frequent total obnubilations. For 12 to 14 hours he had complained of pain back of his left ear and high in his neck.

Examination showed a small, dwarf-like, well formed boy, with hyperkinetic reactions, and a good coat of sun tan. Macewen's sign was positive; circumference of head 49.8 centimeters (nor. 50.2 centimeters); some suboccipital tenderness, without stiffness of neck. Teeth were carious; tonsils were huge ragged bulbs that almost met in the midline; posterior cervical lymph nodes were enlarged. Thoracic contents, spine, genitalia (Fig. 2a), and extremities were not remarkable; abdomen was scaphoid, sigmoid gassy, and the lower pole of the right kidney was palpable. Neurologically, left eye and right eye were 20/66 with considerable concentric contraction of fields (3/2000 disc seen only 8 to 10 degrees from fixation point of Bjerrum screen). Bilateral typical choked disc, with 5.0 diopters elevation but without hemorrhages. Paralysis of right abducens, and complete inability to deviate eyes above horizontal; pupils 6 to 7 millimeters in diameter, with normal reactions; left corneal reflex was slightly sluggish. Minor right facial paresis. Remainder of cranial nerves normal. Speech was spontaneously repetitive of sentence fragments (palilalia), and voice a flat monotone. No disturbance of muscular power, nor of any modality of deep or superficial sensation, to objective testing. Tendon reflexes were equal and active in upper extremities, but knee jerks were absent bilaterally, and 3 to 4 clonic jerks were present at the left ankle; a positive left Oppenheim was present, but no other pathological reflexes. Co-ordination tests showed some dysmetria of the right arm, but no ataxias whatever; minor right dysdiadochokinesia; spontaneous past pointing on the right to the right; considerable hypotonia; marked sway in Romberg position, with listing to the left; gait with normal base, but with veering to the left, and ultimate left fall. Cranial roentgenograms showed sutural diaschisis, and moderately advanced convolutional atrophy; no detectable calcification of the pineal body was present.

The syndrome presented was almost entirely mesencephalic. The supranuclear paralysis of upward movements of the eyes (Parinaud syn-

[1] Presented before the Oregon Pathological Society, Portland, April 23, 1934, and abstracted in Transactions, Northwest Med., 1934, 33:216; also presented before the Staff of the Good Samaritan Hospital, Portland, December 9, 1932.

drome) indicated the region of the anterior quadrigeminal bodies; the dysmetria (without ataxia), the monotonous voice, the veering and late fall, were primarily affections of the anterior tegmentum; the dysdiadochokinesia, past pointing, hypotonia, and absent knee jerks were cerebellar symptomatology due to interference with the superior portions of the brachium conjunctivum embedded in the tegmentum; the clonus, facial paresis, and Oppenheim reflex similarly indicated aberrations of motor projection tracts which here traversed this region. In view of the sudden onset of intracranial tension (vomiting, choked disc, headache, Macewen's sign, sutural diaschisis) a fulminating obstruction of cerebrospinal fluid had occurred; the aqueduct of Sylvius traverses the mesencephalon and is of small caliber. Whether the palilalia and central visceral crises were regarded as thalamic or hypothalamic did not much alter localization. Since objective examination gave no support to symptomatology arising from structures immediately adjoining the mesencephalon, glioma was improbable; age made cholesteatoma, and meningioma of the incisure, unlikely; macrogenitosomia præcox appears in only about 40 per cent of pineal tumors in males, and its absence would not speak against pineal neoplasm. Although lateralization was mostly to the right (clonus, tegmental and brachium conjunctivum signs, abducens, Oppenheim), the opposite side was also indicated (postauricular pain, corneal reflex, seventh paresis); this bilaterality would again favor a lesion lying close to the midline.

A *diagnosis* of pineal tumor was made, and, after the situation and prognosis had been talked over with the boy's parents, he entered the Good Samaritan Hospital September 25, 1932. By this time he was often irrational, and small hemorrhages had appeared about both optic discs; left lagophthalmos was present, with left Oppenheim and Gordon, and right Babinski; he was wholly unable to sit upright or to stand unsupported, falling promptly to the right in both instances; no ataxia or dysmetria was then present; he had apneic periods of 25 to 50 seconds' duration in the course of his Cheyne-Stokes respiration; his weight was only 34 pounds.

In the absence of operative intervention the ultimate prognosis in pinealomas is so hopeless that direct attack upon the growths would seem warrantable. Palliation might be undertaken by subtemporal decompression and roentgentherapy (5), but inasmuch as such tumors sooner or later produce an obstructive hydrocephalus either at the iter or at the tentorial incisure, this procedure must necessarily become wholly ineffective ultimately (33), even if initially possible. In the present instance, with a fulminating internal hydrocephalus already well established, a subtemporal decompression was obviously inapplicable. Suboccipital decompression has uniformly been found promptly fatal, except in the fortunate instance reported by Cairns (12); the mechanism of this latter type of fatality is readily perceptible—in the presence of unalleviated hydrostatic tension anterior to the tumor, release of pressure in the posterior fossa could serve only to impact more tightly any tissue caught within the embrace of the unyielding tentorial incisure, thus compressing mesencephalic centers even further, with consequent decerebrate rigidity, tonic convulsions, intraneoplastic extravasation, and precipitate exitus as sequelæ.

The possibility of direct attack upon the pineal region had been discussed in hortatory generalities in the first decade of this century, but 1913 first saw the clinical problem attacked with a practical determination which soon resulted in an embarrassment of dubious riches. Nassetti's *parasagittal* operation (23) was obviously too mutilating for serious consideration.[1] Krause, having had success in removing a quadrigeminal tumor through the posterior fossa, by traversing *subtentorially* the superior surface of the cerebellum, advocated (25) a similar route for pinealomas but was nevertheless unsuccessful later in extirpating a tumor by this method (16); this exposure is at the bottom of a funnel, is surgically hazardous because of suspensory veins between the cerebellum and tentorium, and furthermore, since most pineal tumors present into the incisure from above, the procedure advances upon the pathological condition from below the impaction; I do not believe the route has since been used. Brunner painstakingly worked out a unilateral *transoccipital* procedure (29), ligating the veins anchoring the occipital lobe to the sinus longitudinalis, retracting the lobe, splitting the posterior portion of the corpus callosum, and transsecting the tentorium alongside the sinus rectus,[2] thus exposing the entire mesencephalic roof. Various minor modifications and supplements of this approach have been used in practically all successful extirpations since that time, as is shown in Table I. The problem thereafter lay dormant until the World War had passed, when Dandy (6), having

[1] Large parasagittal trepanation; resection of sinus longitudinalis, falx, and sinus rectus; bilateral retraction of hemispheres; transsection of posterior part of the corpus callosum, exposing the pineal region. Even if the patient could survive resection of the sinus rectus which, through the great vein of Galen, accomplishes much of the internal venous drainage of the brain, the resection of the posterior part of the sinus longitudinalis would certainly create a diplegia, if not a quadriplegia. In contemporary connection, see the remarkable illustration (Abb. 220) in H. Hoff and L. Schoenbauer's Hirnchirurgie. Leipzig: Deuticke, 1933.

[2] Brunner's original description does not mention the transsection of the tentorium; this important step is first mentioned, so far as I can determine, by Tandler and Ranzi (31) in 1920.

had difficulty with the transoccipital route in experimental animals,[1] modified Brunner's exposure so as to approach the region by a *transparietal* midline route, bisecting the splenium and transsecting the lower part of the falx cerebri; he used this approach to expose one tumor and to extirpate two. In 1931, Van Wagenen (33) published his *transcortical* route, and exenterated a pineal tumor successfully by the procedure.

Four successful extirpations have been accomplished by the Brunner route, one by Dandy's and one by Van Wagenen's. In addition, the Brunner route has been used unsuccessfully (patient not living to be discharged from the hospital) in 4 instances, and in yet another no tumor was disclosed though the operation was carried through successfully and the patient recovered; the route has furthermore been twice used in the same patient to evacuate a gliomatous cyst of the quadrigeminal plate (10, Case 33).[2] Dandy's route has been used unsuccessfully in two instances, and in another the result has not been disclosed.[3]

The detailed late postoperative neurological status of patients surviving the operation is found only thrice in the literature (Foerster, Cairns, Van Wagenen). The ataxias, and the extrinsic and intrinsic oculomotor phenomena which follow transiently, may as readily be attributed to release of mesencephalic pressure as to direct operative trauma; the frequent transient hemiparesis may probably be due either to spreading venostatic edema of the occipital lobe, or to unmentioned direct trauma to the vein of Trolard. In the 2 instances in which vision had not been completely lost before operation, a permanent contralateral homonymous hemianopsia has followed operation—attributed in Cairns' case to the necessitous ligation of the internal occipital vein (which drains the area striata—12), and in Van Wagenen's case to the subcortical transsection of the optic radiation in his approach to the ventricle.[4]

Finally, the modern Brunner approach possesses a still further advantage of almost inestimable value, one which has not heretofore been sufficiently emphasized; this occurs incidentally in the course of the operation by the routine performance of what Naffziger (22) has called an "internal decompression," through transsection of the tentorium alongside the sinus rectus, thus effectively releasing the internal foothold for counter traction theretofore given the tumor, and, in the presence of infiltrative or irremovable growths, offering surest and most prolonged relief from the torture of an obstructive hydrocephalus.

I am aware of no means other than direct operative exploration which can at the present time determine whether a pineal growth be circumscribed or invasive. With the foregoing considerations in mind, the Brunner procedure was chosen for exploration of the pineal region, after ventriculography had given objective confirmation of the clinical diagnosis.

Ventriculography, September 28, 1932, yielded clear cerebrospinal fluid under great tension, but with normal microscopic and serologic findings; the roentgenographic examination showed (Fig. 1) symmetrical internal hydrocephalus without displacement, and a soft tissue mass projecting downward in the midline from the posterior superior portion of the third ventricle. Under procaine anesthesia supplemented by avertin, a left occipital bone flap (Fig. 2 b, c,) was turned down with difficulty, due to increased midline vascularity; the patient's condition became poor; the ventricle was tapped to release air and fluid, and the wound closed. The following day, he had a full blown right ophthalmoplegia externa; the only movement possible in the left eye was a few degrees of downward deviation; both pupils reacted normally, but were obliquely ovoid, and were displaced eccentrically toward the nose. No evidence of hemiparesis; bright, alert, rational, but slept much, with Cheyne-Stokes respiration. In 5 days the left eye had regained normal motion, and the right could perform upward and downward movements. Second stage operation was October 5, 1932; four groups of suspending veins, spanning the space from the sagittal sinus to the occipital cortex, were ligated or coagulated; one of the groups escaped from its ligature and it was ultimately necessary to use muscle stamps from the gastrocnemius to effect stasis; the wound was again closed. There was subsequent anorexia, intermittent periods of marked motor restlessness, and complete unwillingness to talk. By October 15 his weight had dropped to 18 pounds despite high calorie diet, rectal feedings, and intravenous glucose; he was transfused with 225 cubic centimeters of whole blood, and his hemoglobin rose from 65 per cent to 93 per cent.

Third stage exploration was undertaken on October 17, 1932. The occipital lobe was exposed, and, after the final anchoring vein had been doubly ligated with silk, and its tributaries blocked with silver clips, it was divided; the occipital horn was tapped, the lobe protected with saline

[1] It is possible that the bony tentorium in animals was the factor causing his serious difficulties with the veins of Galen.

[2] The route has also been successfully used to biopsy yet another glioma of the quadrigeminal plate, by Foerster in 1930, to my personal knowledge, though the case has not yet been published. It might be added here that Tandler and Ranzi (31) have also advocated this (Brunner) route, *supplemented by a tentorial dural flap*, to expose lesions on the extreme lateral side of the basal mesencephalon—the only known feasible route to this untouchable great cross roads of the nervous system.

[3] In a monograph published since this paper was written (Benign Tumors of the Third Ventricle of the Brain. Springfield, Ill.: C. C. Thomas, 1933, p. 171), Dandy has reported 4 cases apparently using the Brunner approach to tumors lying within the third ventricle, as well as 9 by his transparietal route, and 8 by his transventricular routes. His illustrations of operative procedure would indicate that in the transoccipital route he approaches the region by a dural flap having its pedicle at the midline, and by ligation of the veins along the sagittal sinus on the *inferior-lying* half of the brain, thus allowing gravity to retract the hemisphere and to drain material outward. The position of patient, team, and operator would be awkward, but the surgical advantages undoubtedly considerable.

[4] The extent of the hemianopsia might possibly be diminished in this latter route by placing the cortical incision obliquely horizontal, instead of vertical.

TABLE I.—DIRECT OPERATIVE INTERVENTION IN PINEAL TUMORS

Date	Surgeon	Bibliographic reference	Route	Remarks	Clinical operative result		Sequelæ
					Unsuccessful	Successful	
1913	Brunner, C.	Zur Pathologie und Operabilitaet der Tumoren der Zirbeldruese. H. Rorschach, Beitr. z. klin. Chir., 1913, 83: 451–474	Brunner	Did not disclose tumor	Recovery		Transient paresis of leg, and hemihypesthesia
1914	Pussep, L.	Die operativ Entfernung einer Zyste der Glandula pinealis. Neurol. Zentralbl., 1914, 33: 560	Brunner	Evacuation of cyst	Died few hours later		
1921	Dandy, W. E.	An operation for the removal of pineal tumors. Surg., Gynec. & Obst., 1921, 33: 113–119	Dandy	Infiltrative tumor of cerebellum; no attempt at extirpation	?		
	Ibid.		Dandy	Removal of hard nodular tuberculoma of pineal, 4 by 5 cm.		Died 8 mos. later, presumably of other tuberculous lesions	
	Ibid.		Dandy	Cerebellar op. 10 days antecedent; extirpation of 26 gm. pinealoma with 7 cm. of embedded veins of Galen	Died in shock 48 hr.		
1925	Cushing, H.	Pineal pathology: further studies. G. Horrax and P. Bailey. Arch. Neurol. & Psychiat., 1928, 19: 394–413, (Case 1)	?	Shown by ventriculography, but not located at operation; necropsy showed exploration of third ventricle anterior to growth	Died few days later		
1926	Sachs, E.	Diagnosis and Treatment of Brain Tumors. St. Louis: C. V. Mosby Co., 1930, p. 276	Modified Brunner	Cystic tumor removed, size of plum, ?cholesteatoma		Alive 7+ weeks	Meningismus necessitating laminectomy 3 weeks after operation; neurol. status not given
1926	Krause, F.	Operative Freilegung der Vierhuegel, nebst Beobachtungen ueber Hirndruck und Dekompression. Zentralbl. f. Chir., 1926, 53: 2812–2819	Krause	?Pinealoma; exploration, but unable to remove	Recovery		
1927	Foerster, O.	Das operative Vorgehen bei Tumoren der Vierhugelgegend. Wien. klin. Wchnschr., 1928, 41: 986	Brunner	25 gm. tumor 4 cm. in diameter, probably endothelioma		Alive 28+ months	Eyes deviated right 48 hours; blind of 2° atrophy 4 months before operation. No persistent sequelæ; brilliant result
1931	Van Wagenen, W. P.	A surgical approach for removal of certain pineal tumors. Surg., Gynec. & Obst., 1931, 53: 216–220	Van Wagenen	Transcortical transventricular exenteration of non-invasive pinealoma		Alive 15+ months	Transient left hemiparesis; Biot respiration with CSF block 48 hours. Minor left hemihypesthesia and astereognosis; permanent l. homon. hemianopsia
1932	Cairns, H. W. B.	Diagnosis and treatment of pineal tumors. W. Harris and H. W. B. Cairns. Lancet, 1932, 1: 3–8	Brunner	Negative cerebellar op. 6 mo. antecedent. Total extirpation of 28 mm. circumscribed pinealoma		Alive 14+ months	Eyes deviated rt. 48 hours A-R pupils. Left hemiparesis 6 wks. Left astereognosis and bathyesthesia. Permanent l. homon. hemianopsia
1932	Allen, S. S.	Tumors of the third ventricle. S. S. Allen and H. W. Lovell. Arch. Neurol. & Psychiat., 1932, 28: 990–1006, (Case 6)	Brunner	Pinealoma 1.5 cm. diameter; uneventful convalescence		Alive 30+ months	Leptosomatic; unable to maintain upward deviation of eyes

TABLE I.—DIRECT OPERATIVE INTERVENTION IN PINEAL TUMORS—Continued

Date	Surgeon	Bibliographic reference	Route	Remarks	Clinical operative result		Sequelæ
					Unsuccessful	Successful	
1932	Allen, S. S.	Ibid., Case 7	Brunner	Adenocarcinomatous teratoma, size of walnut	Died 2½ months post-op.		Stupor for 14 days; left hemiparesis; improvement, but sudden failure. Post-mortem: softening of quadrigeminal lamina and marked cerebral edema
1933	Bailey, P.	Intracranial Tumors. pp. 333–336. Springfield, Ill.: C. C. Thomas, 1933, pp. 333–336	?Dandy	3 by 7 cm. teratoid, oblique from lateral recess to occipital lobe; medial third removed by bilateral operation*	Survived 9 days		Pre-operative "decerebrate"; status ameliorating after operation
1934	McLean, A. J.		Brunner	Circumscribed parapineal teratoma	Died in few hr.		

*Described as (personal communication) "consisting mostly of connective tissue, with islands of epithelial cells sometimes forming cysts." The situation and histological description of this teratoid would seem to make it not unlikely that it arose from remnants of the fiber of Reissner, which except in normal adult humans, extends from the subcommissural organ to the filum terminale of the cord through the central canal (15).

cotton and gently retracted upward from the falx and tentorium by a broad ribbon retractor, thus exposing the straight sinus and the vein of Galen (Fig. 3). The brain stem was found tightly compressed within the incisure of the tentorium. The presenting posterior 2 centimeters of the splenium were transsected, with subsequent persistent escape of clear cerebrospinal fluid; no sign of tumor. The tentorium was incised outward for 3.5–4.0 centimeters alongside the sinus rectus, up to the point of two large transverse venous tributaries. When the tip of this flap was retracted upward, the exposed arachnoid cistern overlying the pineal region was seen to be gray and thickened; the portions nearest the junction of the vein of Galen and the straight sinus bore a number of grouped, pin-head sized, whitened thickenings, which resembled arachnoid tubercles. The arachnoid was incised, exposing both large lateral tributaries of the great vein of Galen. Underneath this lay still another arachnoid layer, and when this latter was also incised there lay exposed (Fig. 3) the left lateral pole of a comparatively smooth edematous, grayish-yellow tumor, estimated to be about 28 millimeters in diameter. The flattened left branch of the vein of Galen was adherent to this mass and was dissected free with scissors without injury to the vein. Traction on the exposed pole of the tumor, with pituitary rongeurs, showed it probably movable. A portion about a half centimeter in diameter was removed for biopsy, and the dissection continued medially. Inasmuch as the tumor seemed to deliver well, traction was continued and a further segment, probably representing a third of the total tumor was thus obtained. These test extirpations showed practically no bleeding and had disturbed the patient but little; though his blood pressure was now reported as unobtainable, his pulse remained at 140 and his respirations were good. Gentle intermittent traction was applied to the remaining portion of tumor, and some final dissection done with patties about the two lesser veins of Galen, which were stretched bloodlessly tight over the top of the tumor. As cautious tractive pull was carried directly laterally, a rounded mass about 18 millimeters in diameter was slowly delivered from under the intact taut veins (Fig. 4). During the latter part of this delivery the patient had an oculogyric crisis and his respirations stopped for several seconds, but were quickly resumed spontaneously. There was a minimal amount of venous bleeding from the bed of the tumor on the right, but this was readily controlled by placement of warm saline patties; total amount of bleeding here was probably not more than 3 to 4 cubic centimeters; the tumor bed was left entirely dry; the collapsed occipital lobe lowered into place, floated up with warm saline, and the wound closed in the usual manner during the next 55 minutes. Despite stimulants (caffein, adrenalin) and glucose, his blood pressure remained unobtainable in the brachial artery, though his femoral pulse remained constantly regular at 120 per minute and his respirations 22 per minute.

An hour and a half after delivery of the tumor, and as final scalp sutures were being placed, his respirations were 12 per minute and labored, though femoral pulse was still 120 per minute. Despite intravenous lobelin, artificial respiration, warmth, and intracardiac adrenalin, the patient's heart action ceased at 4:20 p.m., 1½ hours after completion of closure, and 3 hours after delivery of the tumor.

Complete necropsy was obtained (G. S. Path. 865); there were no significant somatic changes; careful microscopic studies were made of pituitary, thyroid, thymus, adrenal, pancreas, and testis, without detection of any departure from normal. The brain was fixed in 10 per cent formalin and examined; the operative field was in good shape; the occipital lobe showed no gross evidence of trauma; and the tumor bed was without hemorrhage, though tissues were slightly stained (Fig. 5). Hydrocephalic dilatation and atrophy existed only anterior to the entrance to the Sylvian aqueduct. A normal but flattened pineal body 3 by 4 by 6 millimeters in diameters was found (Fig. 6) pushed downward and backward to the right, lying in apposition and just lateral to the

TABLE II.—REPORTED PINEAL TERATOMAS

Year	Author	Age	Sex	Reference
1875	Weigert, F.	14	M	Zur Lehre von der Tumoren der Hirnanhaenge. Arch. f. path. Anat., 1875, 65: 212–219.
1879	Falkson, R.	16	M	Ein Chondrocystosarcom im dritten Ventrikel. Arch. f. Path. u. path. Anat., 1879, 75: 550–553.
1887	Coats, J.	13	M	An adenoid sarcoma with cartilage originating in the pineal gland. Tr. Path. Soc. Lond., 1887, 38: 44.
1889	Gauderer, L.	12	M	Zur Casuistik der Zirbeldruesentumoren. Inaug. Diss., Giessen, 1889.
1896	Gutzeit	8	M	Ein Teratom der Zirbeldruese, Koenigsberg, Diss., 1896.
1899	Ogle, C.	16	M	Sarcoma of the pineal. Tr. Path. Soc. Lond., 1899, 1:4.
1899	Oestreich and Slawyk	4	M	Riesenwuchs und Zirbeldruesengeschwulst. Arch. f. path. Anat., 1899, 157: 475.
1900	Neumann, P.	27	M	Ein neuer Fall von Teratom der Zirbeldruese. Inaug. Diss., Koenigsberg, 1900.
1909	Frankl-Hochwart	5	M	Ueber Diagnose der Zirbeldruesentumoren. Deutsche Ztschr. f. Nervenh., 1909, 37: 455.
1911	Bailey and Jelliffe	12	M	Tumors of the pineal body. Arch. Int. Med., 1911, 8: 851–880.
1913	vandenBergh and van Hesselt	8	M	Tumor glandulae pinealis sive epiphysis cerebri. Nederl. Tijdschr. v. Geneesk., 1913, 1: 1271.
1913	Hueter	19	M	Teratom der Zirbeldruese. Muenchen. med. Wchnschr., 1913, 60: 895.
1913	Takega	10	M	Die Erkrankungen der Epiphysis. Nisshu Igaku, 1913, vol. 3.
1914	Fukuo	19	M	Ueber die Teratome der Glandula pinealis. Inaug. Diss., Muenchen, 1914.
1914	Schmincke	19	M	Ueber die Teratome der Zirbeldruese. Muenchen. med. Wchnschr., 1914, 61: 2043.
1914	Ibid.	22	M	Ueber die Teratome der Zirbeldruese. Muenchen. med. Wchnschr., 1914, 61: 2043.
1915	Odermatt	10	M	Zur Diagnostik der Zirbeldruesentumoren. Inaug. Diss., Zurich, 1915.
1919	Boehm, E.	9	M	Zirbeldruesenteratom und genitale Fruehreife. Frankf. Ztschr. f. Path., 1919, 22: 121.
1920	Frank	20	M	Epiphysentumor bei einem 20-jaehrigen Mann. Berl. klin. Wchnschr., 1920, 57: 888.
1921	Klapproth, W.	15	M	Teratom der Zirbel, kombiniert mit Adenom. Zentralbl. f. allg. Path. u. path. Anat., 1921, 32: 617.
1921	Luce, H.	5	F	Zur Diagnostik der Zirbelgeschwuelste und zur Kritik der cerebralen Adipositas. Deutsche Ztschr. f. Nervenh., 1921, 68: 187.
1925	Horrax and Bailey	7	M	Tumors of the pineal body. Arch. Neurol. & Psychiat., 1925, 13: 439–441. (Case 6.)
1928	Idem.	9	M	Pineal pathology; further studies. Arch. Neurol. & Psychiat., 1928, 19: 394-413.
1932	Allen, S. S., and Lovell, H. W.	19	M	Tumors of the third vertricle. Arch. Neurol. & Psychiat., 1932, 28: 990–1006. (Case 7.)
1933	Altman	11	M	Ueber ein Dermoid der Zirbeldruese. Wien. klin. Wchnschr., 1930, 43: 108–111.

right superior quadrigeminal body. The posterior commissure was intact; the habenular commissure was torn in its central part; all veins of Galen were intact.

The *tumor* was a firm, slightly knobby, smooth surfaced, grayish yellow mass 21 by 18 by 26 millimeters in diameters, to which were adherent on its upper and posterior surface a few elongated shreds of tela choroidea which extended to embed themselves in the body of the growth on its posterior-inferior flattened rugous surface, after stretching over three-quarters of the tumor's circumference. The surface shown by section of the Zenker-fixed specimen was firm, and exhibited numerous minute cysts scattered heterogeneously about, varying from pinpoint size to 1.5 millimeters in diameter; texture varied considerably; macroscopic calcification was not found. *Microscopic* examination showed a remarkable array of tissue (Fig. 7): many of the cysts were lined with stratified epithelium which overlay definite corium; beneath these were sweat glands, sebaceous glands, areolar, elastic, and connective tissue, intermingled with smooth muscle; no hair follicles or hair was found; other small cysts were lined with columnar epithelium, and some with ciliated epithelium; definite bronchial mucosa and small bronchial tubes were found, the surrounding tissue here being composed of areolar, adipose, and connective tissue intermingled with generous amounts of non-branching strands of striated muscle, and islands of cartilage; scattered formations of cartilage in all stages of development were found throughout the tumor, together with occasional islands of osteoid tissue, but no cortical bone or marrow was found; a few collections of calcospherites were present; two areas bearing resemblance to an early enamel organ were found. Choroidal plexus tufts, with good blood supply, had been carried deeply into a nearly-closed small cavity on the posterior surface of the tumor. Glial tissue was not uncommon both in the periphery and within the body of the tumor, but no tigroid substance was found; occasional nests of ganglion cells and rare non-medullated fibers were present in relation to the periphery of glial areas within the mass of tumor; not only were pseudo-rosettes often found, and fully differentiated ependymal lining to at least one small cyst, but multiple ependymal tubulization was occasionally present within the glia feltwork, as is found in congenital or traumatic atresia of the aqueduct. Vascularization was not abundant, but no areas of necrosis were present.

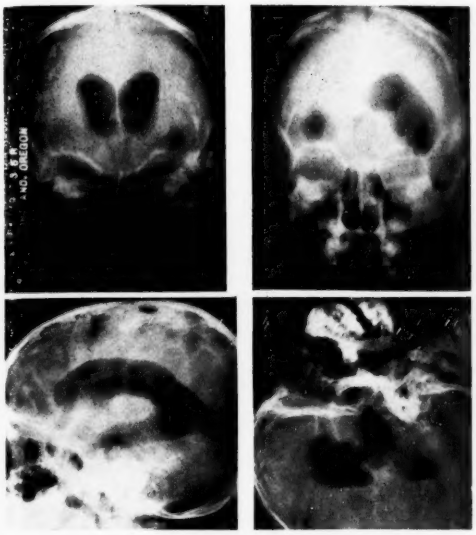

Fig. 1. Ventriculograms. a, An anteroposterior projection showing uniform symmetrical hydrocephalus of lateral and third ventricles. b, A postero-anterior projection showing undisplaced lateral ventricles, with hydrocephalic third ventricle containing a rounded mass posteriorly projecting into it from above. c, One film of a lateral stereoroentgenogram showing convolutional atrophy, with hydrocephalic dilatation of lateral ventricles and of foramina of Munro; the anterior part of the third ventricles is faintly demonstrated. d, A view showing filling defect in posterosuperior portion of third ventricle; air is in both frontal and temporal ventricular horns, and in third ventricle.

Fig. 2. Patient 2 days before third-stage operation. a, shows emaciation and normal genital development. b and c are views of the occipital wound after two interventions.

This teratoma has a more complicated structure than any other described in literature available to me; structures derived from all three germ layers are indubitably present. If a teratoma (as distinguished from a dermoid) be a tumor comprised of tissue arising from more than one germinal layer, many recorded pineal teratomas are entitled to their designation only by virtue of small islands of cartilage or of smooth muscle within or about a tumor composed predominantly of true pineal parenchyma in some stage of differentiation[1] (13); while others described in the literature as "dermoid" (e.g., 2[2]) actually contain at least epidermal and mesenchymal structures, and should be denominated teratoma. The problem of identification, however, is not so uncomplicated as would at first appear, for connective tissue is present in pineal trabeculæ, the gland is invested with pia, has a neural pedicle, and, at least in certain vertebrates (calf, ox), smooth muscle and striated muscle (24) are normally present; human pineal tumors containing fibers of striated muscle in their capsules have been excluded on occasion from teratomal classification by some authors (26). In gliomas, Bailey and Cushing recognized the logic of accepting the predominant type of tissue present as indicative of tumor name and tumor behavior; if such a practical rule were adopted for reported pineal teratomas, several could be excluded from Table II.

It is probable furthermore that not all tumors reported as pineal teratomas actually arise from the pineal body itself. It is true that teratomas may actually arise from within pineal substance, and that pineal tissue is then found embedded in the walls of the tumor (2), but Ewing (8) mentions that in cases of "pineal teratoma" a normal uninvolved pineal body is sometimes found; moreover, in the case under report a flattened pineal body of normal structure for the patient's age was found pressed backward and downward onto the right anterior quadrigeminal body and free underneath the tumor itself. Teratomas, of course, may occur elsewhere within the cranial cavity than in the region of the pineal (parahypophyseal, occipital, etc.) and dermoids—often containing ciliated epithelium (17)—are not uncommon throughout the midline craniospinal axis, but one enters an exceedingly fascinating field in tracking down possible origins for what may be termed para-pineal teratomas, such as the one herewith considered.

[1] Such tumors as these might strictly be denominated teratoids.

[2] Contained corium, squamous epithelium, hair, cholesterin, sebaceous glands, adipose tissue, cartilage, lamellar bone, marrow, osteoid tissue, et .

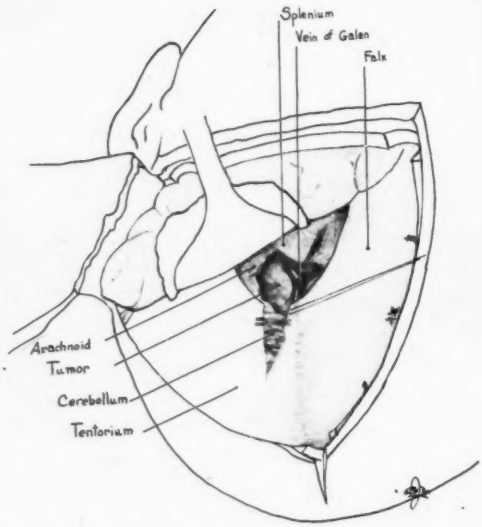

Fig. 3. Operative field; drawing is elaborated from immediate postoperative sketch. The exposure might be further facilitated by incising upward the inferior border of the falx, but the additional mobility thus given the great vein of Galen augments its chances of fatal rupture during removal of the tumor.

POSSIBLE ORIGINS

A not uncommon origin for complex tumors is from embryonic vestiges. The human third ventricular cavity is remarkable embryologically for the numerous outpouchings to which it gives rise, and which ultimately come to form special organs —pallium, optic vesicles, infundibulum, postinfundibular recess (28), choroid plexus, paraphysis, pineal, and subcommissural organ (15); in addition, there occur in embryonic life a parapineal process and two precommissural organs. To quote from Wilder (34):

Fig. 4. Photograph of the Zenker-fixed parapineal teratoma removed at operation.

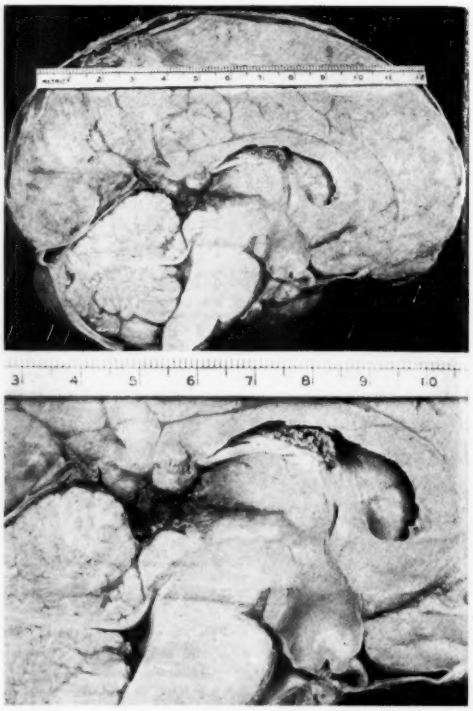

Fig. 5. Above, Median sagittal section of brain, showing left half of tumor bed. Below, The condition of the operative field and the comparative lack of regional trauma possible with the Brunner operative procedure; note the intact posterior commissure and the ruptured habenular commissure.

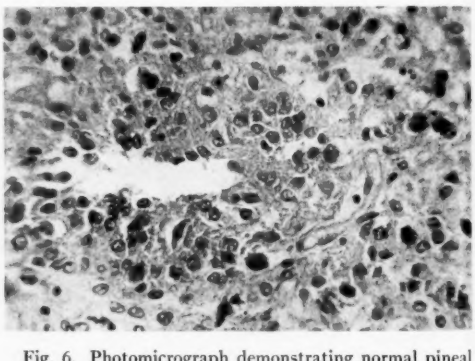

Fig. 6. Photomicrograph demonstrating normal pineal parenchymal histology in the somewhat flattened pineal body found pressed downward and to the right by the overlying parapineal teratoma.

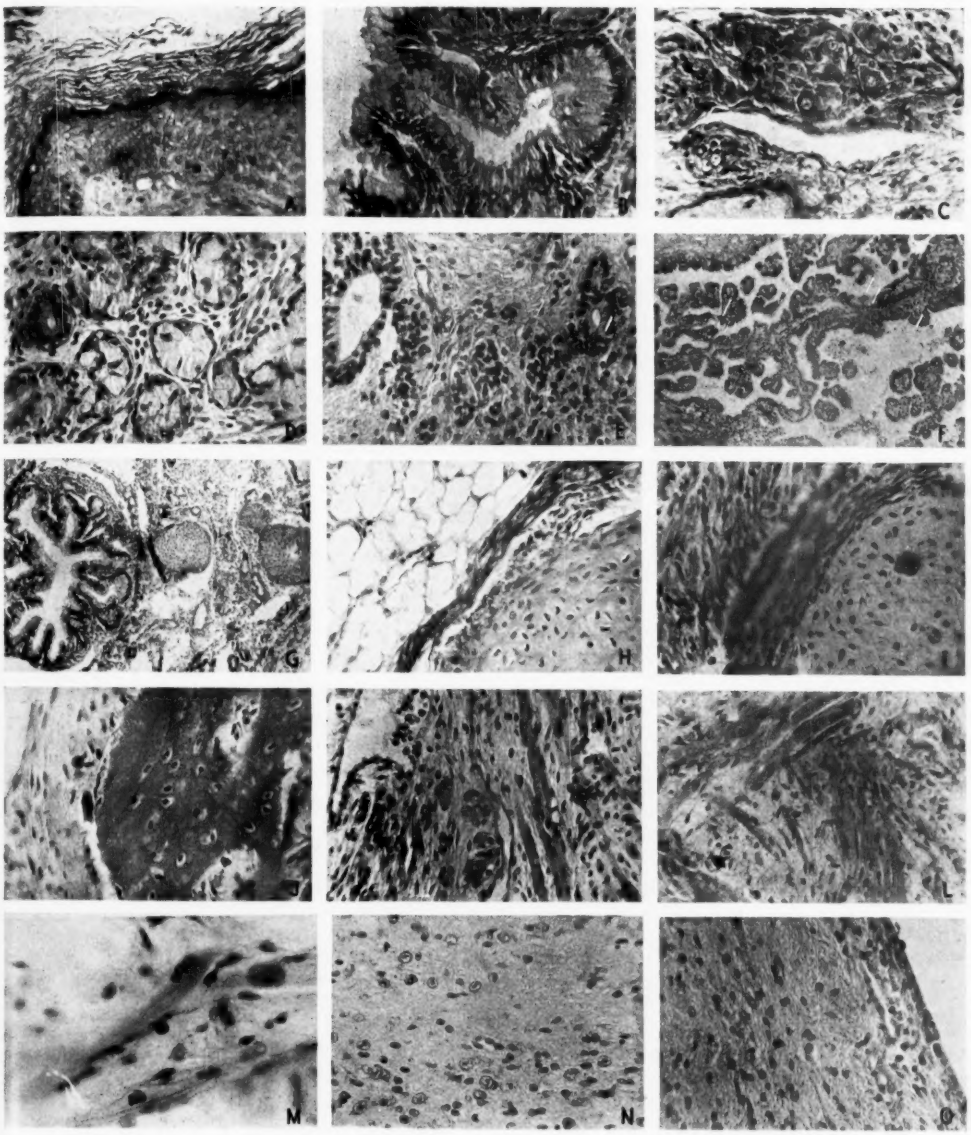

Fig. 7. Photomicrographs showing the variety of tissue comprising the teratoma. a, Corium and cornified layer of squamous epithelium; b, ciliated epithelium; c, nest of ganglion cells at edge of glial island—a blood vessel is in the lower left field; d, sebaceous glands; e, ependymal tubulization of marginal glia; f, choroid plexus; g, small bronchus with cartilage islands, and areolar tissue; h, adipose and connective tissue, with segment of island of cartilage; i, calcospherite in cartilage; j, osteoid tissue; k, smooth and striated muscle near bronchiole; l, interlacing striated muscle—note abundance of cross striations; m, oil immersion of cross striations, shown best in oblique fiber near top of field; n, astrocytic feltwork in body of tumor; o, adult ependymal lining of microcyst.

Several of these (outpouchings) occur along the dorsal aspect, where in addition to the choroid plexus of the third ventricle, there appear in the thin roof typically three median diverticula, which, owing to the varying grades of development which they show in different animals, have been more or less confounded with each other. The most anterior of these is the paraphysis. . . . Posterior to the velum transversum there are two possible diverticula in the diencephalic roof, both correctly termed epiphyses, parietal organs, or pineal organs. Both show a tendency to pass through the skull and attain a position directly beneath the skin in the middle line, developing there a rudimentary sense organ of uncertain nature but probably an eye in each case. The identity of the structure may always be determined from its central connections.

In the cyclostomes, fishes, and reptiles both epiphyses are usually present and one or the other may attain a high degree of development, in some cases reaching its height during embryonic life, and in others appearing as a well-formed organ in the adult. Thus in the cyclostome *Petromyzon*, the optical nature is indicated by the presence of pigment in what may be a vestigial retina. In the selachians, the epiphysis passes through a minute foramen in the skull and reaches the surface, where its terminal organ is visible. The highest development is reached among certain lizards (e.g. Hatteria punctata[1]) where the terminal organ lies in a socket, the parietal foramen, formed in the interparietal suture, and represents a fairly good eye with pigmented retina, a more or less makeshift lens, and a well developed nerve connecting the terminal organ with the brain. Above this on the surface, is situated a transparent scale surrounded by a ring of smaller opaque ones, making a conspicuous object on the heads of these forms. In birds and mammals the epiphysis is reduced to the form of the so called "pineal-gland" pushed backward from its original position (and deeper) by the growth of other parts.[2]

In human development almost all of these diverticula, which sometimes indeed become racemose, are lined with some variety of cuboid or columnar epithelium analogous to ependyma, and may at times attain cilia upon their free surfaces (32); reported tumors of the adult human paraphysis are remarkable for their simple histological structure (27, 11); complexity of structure in such third ventricular diverticula is encountered developmentally only when apposition to ectoderm is again attained (eye, hypophysis, parietal eye). Alexander has described a mid habenular nodule in 25 per cent of human embryos (1), corresponding to the mammalian corpusculum parietale (Krabbe), a possible vestige of the more anterior epiphysial diverticulum first described in human beings, I believe, by Mihalcovics (1877).[3] I am unable to ascertain definitely whether the tumor under report arose from one of the lateral paired precommissural organs (32), or from the mid habenular corpusculum parietale, but, from macroscopic examination, incline to the latter view; the internal histological structure of the corpusculum is unknown to me. The comparative and human embryology (and histology) of the pineal body itself affords no utilizable clue for the development of teratomas. A continuing uncomplicated proliferation of any of the four vestigial structures of this region (corpusculum, pineal, precommissural organs) is therefore insufficient to explain a trigerminal layered tumor.

However, since most of the diencephalic roof structures are midline, or immediately abut it, Cohnheim's inclusional theory might operate here with peculiar force, and, through the enfolding of epithelial vestiges, could explain not only the regional dermoids, lipomas, cholesteatomas, and colloido-mucinous cysts, but also by more primitive inclusion of totipotent blastomeres, even account for the occurrence of teratoids and teratomas. The stimulus to proliferation would be, under this theory, somewhat more obvious here as a continuing process than in the often delayed development of most other parenchymal neoplasms—for indeed 75 per cent of pineal teratomas are found to occur in pre-adolescents (Table II), their crucial relation to the tentorial incisure causing them early to become manifest.

Another possibility remains to be considered: that of pseudogestation of a fertilized filial polar body[4]; Askanazy indeed has even reported a primary chorio-epithelioma of the pineal body. This possibility might have had peculiarly effective force until 1921, for until that year all reported teratomas had been found solely in males[5]; Luce, however, at that time reported an undoubted pineal teratoma which had occurred in a 5½ year old girl (18). Of course, to confirm a Mendelian male sex-linked character pineal teratomas would have to be demonstrated as occurring in alternate generations of males, transmitted through unaffected females; such demonstration has not been brought forward. Ferguson, however, has recently demonstrated presence of prolan A in the urine of males (9) with teratoma testis[6]; whether

[1] See Dendy, A. On the structure, development, and morphological interpretation of the pineal organs and adjacent parts of the brain in the Tuatara (Sphenodon punctatus). Tr. Roy. Soc. Lond., 1910, June.

[2] These statements are based principally, I believe, on the works of Marburg (19), Edinger (7), and Krabbe (14). Wilder makes no mention of the remarkable series of diverticula which arise from the dorsal sac in the roof of the third ventricle of some ganoid fishes; these are described by Edinger (pp. 205–206) as nearly encircling the brain stem, and, from descriptions, it may be that the rudimentary human precommissural organs are the analogue of these structures.

[3] Der Recessus suprapinealis ist durch eine secundaere Aussackung nach oben und hinten entstanden . . ., p. 100 (21).

[4] Parthenogenetic development of an adherent included polar body would come within scope of this category as well.

[5] If pineal teratomas were found to occur solely in males, one might suspect some partitional aberration of chromosomes whereby the discarded polar body (female chromosomal) could regularly give rise to teratomal malformations.

[6] Found also in chorio-epitheliomas of either sex, and of course in normal pregnancy; the amount of prolan A in cases of teratoma testis with adult type tissues (cartilage, respiratory epithelium, etc.) was found to be much less than in tumors composed of more embryonic tissue; it is probable therefore that even if prolan A had been present in D. F.'s urine the amount might have been undetectably small.

this be a reaction common to all teratomas is yet unknown. Moreover, Silberstein and Engel believe that they have demonstrated an estrogenic substance in the normal beef pineal body. In the present case no tests for prolan were made, illuminating though they might have been; indeed, I was delighted to confirm operatively a clinical diagnosis of tumor of pineal region, let alone to attempt polishing the diagnosis by prediction of the precise histological characteristics of the neoplasm.

Since there is no conceivable possibility of semen coming in contact with the pineal or with primitive inclusional ova dormant there, it is not necessary to consider non-coaetaneous generation, as one inevitably must in ovarian or testicular teratomas.

SUMMARY

A case of parapineal teratoma is recorded; this is the first removed operatively; the operation was not clinically successful. Operative procedures, tumor classification, and possible origins of the tumor are considered.

Grateful acknowledgment of criticism and aid is made to Drs. Charles Manlove, Albert Mathieu, Harry Blosser, Matthew Riddle, Louise Eisenhardt, and George Snyder; Miss Wanda Templeton assisted in the histologic work.

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LOSS OF URINARY CONTROL ASSOCIATED WITH RELAXATION OF THE VESICAL NECK

A Modified Technique for Its Treatment[1]

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INCONTINENCE of urine is frequently associated with the trauma of childbirth or some sort of dystocia. The degree of the patient's difficulty ranges from the loss of small amounts of urine upon coughing or sneezing to complete incontinence with constant soaking of the patient's clothing with urine. This is often, but not always associated with cystocele and relaxation of the urethral floor. Cystoscopic examination usually reveals a symmetrical deformity of the sphincter at the vesical neck showing a defect at the posterior or vaginal side of the sphincter (8). Operative treatment alone is of any avail.

A large number of methods have been proposed for the treatment of urinary incontinence of this type (1, 3, 10, 11, 12), and have been employed with varying success. The Kelly method has been the most successful procedure in this type of incontinence and is usually sufficient to cure the average case of relaxation of the vesical neck. The anatomical and functional relationship of the internal and external sphincters and the importance of the fascial support have been very adequately discussed in the papers of E. L. Young, Bonney, Watson, and others. There are, however, certain cases with marked thinning of the bladder wall at the vesical neck, i.e. the site of the internal and external sphincters (usually indistinguishable from one another at operation), in which it is very difficult to implicate the relaxed tissues of the redundant vesical neck sufficiently (about a Pezzer catheter) to insure the competency of the sphincter. The difficulty lies in the fact that only very small superficial sutures may be taken in placing the mattress sutures employed in the Kelly technique and, unless extreme care is employed, there is danger of penetrating the vesical mucosa, with the subsequent danger of the development of a fistula, a most troublesome and embarrassing accident.

These considerations have resulted in several failures of complete functional cure following the Kelly technique, presumably due to the fact that the plicating stitches were placed too superficially, and this in turn due to the fact that there seemed to be no muscular or fascial structure worthy of the name to suture adjacent to neck of the bladder. The plan suggested itself whereby the tissues surrounding the vesical neck might be thickened sufficiently to permit the plicating sutures to be passed more boldly and deeply, imbricating more tissue. This was effected by the employment of superimposed pursestring sutures originally employed, by Gersuny and Saenger, to reduce the bladder wall in large attenuated cystoceles. The prime purpose of this procedure, however, is to insert a valve-like bunching of tissue at the lower margin of the vesical neck.

This method was devised after two unsuccessful attempts to cure a case of relatively simple postpartum incontinence. This patient was seen first on November 23, 1933.

She was a primipara, white, aged 24 years, who had been subjected to a complicated breech extraction procedure 6 months previously and since then had had complete diurnal urinary incontinence.

On examination the outlet was found to be moderately relaxed. The anal sphincter was in good condition. The cervix showed a laceration with a moderate erosion. There was a fairly large cystocele, the bladder coming down low on the cervix. There was rather marked pouching under the urethral orifice but this was proved by sound not to be the urethra. The fundus was in retroversion, readily movable. It was normal in size and consistency.

Operation: A median longitudinal incision was made, the bladder mobilized and advanced. A Pezzer catheter was placed in the bladder and Kelly mattress sutures were placed plicating the neck of the bladder anterior to the "drag" of a Pezzer catheter which was withdrawn before the sutures were tied. This narrowed the vesical neck rather markedly. Anterior colporrhaphy was done, the fascia being sutured in a separate layer. The urethra was mobilized and advancement of the urethra was performed after the manner of Pawlik, a horseshoe denudation being made above and lateral to the urethral orifice. The defect was closed with interrupted chromic catgut sutures. The vesical mucosa was sutured with interrupted sutures of chromic catgut No. 1. The patient was catheterized frequently and her urinary control was much improved but was not complete, even during her stay in bed. On discharge from the hospital her control was further improved but there was still considerable intermittent leakage unless she emptied her bladder very frequently.

The patient was dissatisfied with the result and was next operated upon April 17, 1934. On examination there was evidence of a cystocele repair well healed. The urethral orifice was rather dilated and seemed large on passage of a sound. The uterus was in midposition and was freely movable. There were no lateral masses. The patient had a diurnal urinary incontinence only. Generally, she retained her urine perfectly at night if she got up to void once or twice.

Second operation. A Pezzer catheter was placed in the bladder and the vesical neck was demonstrated at quite a

[1] From the Department of Obstetrics and Gynecology, Western Reserve University School of Medicine, and the Lakeside Hospital.

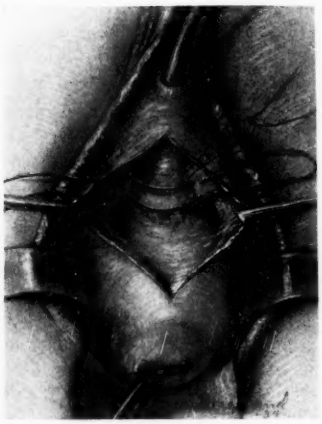

Fig. 1. Diagrammatic view of pursestring *in situ* showing sutures placed in the vesical neck.

high level. The urethra was dissected out and Kelly mattress sutures of chromic catgut No. 1 were placed in the musculature above and below and deeply into the fascia overlying it. The urethra was covered further by two interrupted sutures narrowing it anteriorly toward the urethral orifice. Several interrupted sutures were placed in the fascia posterior to the vesical neck, giving the bladder and presumably the sphincter mechanism additional support. The mucous membrane and fascia were closed in one layer with interrupted sutures of catgut No. 1. This material was used in very small round needles throughout. Patient was catheterized every 6 hours and a retention catheter was not employed at any time. The patient was kept in bed for 2 weeks and was discharged from the hospital with her condition essentially unchanged. Her urinary control was improved, but there was slight and constant leakage. After her return home, her condition failed to improve. Unfortunately, it is unusual for urinary control to improve after discharge from the hospital. More frequently, at a later date, incontinence may recur following an apparently successful operation. In view of the disappointing result it seemed advisable to attempt something else in an effort to restore the competency of the vesical sphincter mechanism. It was decided to attempt to produce a valve or obstruction at the vesical neck.

Third operation. A median incision was made through rather dense scar tissue overlying the bladder and the mucosa was carefully mobilized, leaving a rather thin fascial investment covering the vesical musculature.

Two superimposed pursestring sutures approximately 2 centimeters in diameter were placed just anterior to the vesical neck as demonstrated by the "pull" of a Pezzer catheter. The first suture was invaginated and tied (Fig. 1), after the manner of Gersuny and Saenger, and a second pursestring suture was then placed immediately over the first suture and likewise invaginated. This narrowed the bladder from side to side considerably. Two Kelly mattress sutures were placed immediately over the superimposed pursestring sutures (Fig. 2). The Pezzer catheter was withdrawn, being carefully stretched over a stylet before the mattress sutures were tied. Patient was returned to the ward and was catheterized every 6 hours for 48 hours.

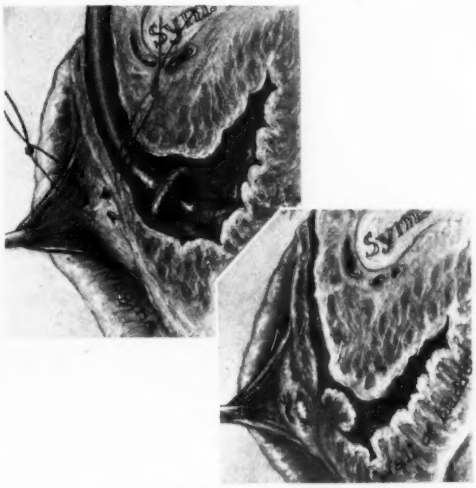

Fig. 2. Above, Sagittal section showing position of pursestring and Kelly mattress sutures. Below, Sutures tied showing invagination of thinned out vesical neck.

She started voiding spontaneously after this time and was entirely continent from the date of operation. She had not the slightest leakage at any time in bed. She was completely continent when she got up on her fourteenth hospital day. She, however, had slight leakage upon coughing. Cases of an attenuation or atrophy of the musculature structures surrounding the vesical neck may be susceptible of attack by this rather simple device. It suggests the production of something analagous to a median bar in the prostate represented diagrammatically in Figure 2.

We have not had the opportunity of inspecting the vesical neck since the operation but cystoscopy will be done 3 months after operation. It is dangerous to subject a recently repaired sphincter mechanism to the dilatation incident to cystoscopy. It is quite possible that the invaginated tissue inside the pursestring sutures may atrophy and control subsequently may not be so complete as at present. However, at the present time, 2 months after operation, the "plug" mechanism, collapsing the urethra from below at the vesical neck, is functioning well.

The success of this simple procedure restoring competency of the vesical sphincter mechanism in

a case which had resisted two attempts at repair in a conventional manner seemed quite striking. It is obviously best adapted to cases in which there is no true evulsion or destruction of sphincters, but merely its loss of function through having been stretched or markedly attenuated.

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THE SIGNIFICANCE OF BLEEDING OR DISCHARGE FROM THE NIPPLE[1]

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EDUCATIONAL propaganda for the prevention of cancer is responsible for more women seeking surgical advice than ever before. Bleeding or a discharge from the nipple is not infrequently one of the symptoms that causes an anxiety on the part of a patient. This symptom is recognized by surgeons and pathologists as indicative of pathology in the mammary gland but unfortunately its significance, as well as its bearing on treatment, causes conflicting opinions.

Topie states that, in a few cases, loss of blood from the nipple results from benign intracanalicular papillomatous growths but most frequently the blood results from an endocanalicular dendritic carcinoma. Adair is of the opinion that a dark, bloody discharge always is indicative of a ductal carcinoma. Deaver and McFarland interpret blood from the nipple as being attributable in the majority of cases to papillary growths or to benign intracanalicular papillomas. Lewis believes a serohemorrhagic or brownish discharge is not a clue to malignancy but is associated with intracanalicular papillary cystadenoma and the adenocystic type of chronic mastitis. Saar considers that there is no relationship between a bleeding from the nipple and carcinoma. Greenough and Simmons report 20 cases of cystadenoma, in 11 of which there was a discharge from the nipple and 3 of these were found to be malignant. Rodman regards cystadenomas situated immediately behind the nipple with a discharge of blood in patients 48 to 49 years of age as being potentially malignant from their inception. Bloodgood found that less than 1 per cent of his cases of carcinoma of the breast had bleeding from the nipple. His practice is to remove every palpable tumor in the breast regardless of the discharge from the nipple and to make a diagnosis of the tumor by immediate frozen section at operation. His studies reveal that a patient with a lump in the breast and a discharge from the nipple which has not been noticed longer than a month has an 80 per cent chance of having a benign lesion. Following a recent review of his cases with discharge from the nipple that have not been operated, he feels convinced that these patients run no more risk of developing cancer than does a woman with no discharge (9).

Cheatle and Cutler, in their recent book, *Tumors of the Breast* differ in opinion with Bloodgood. They discard the old nomenclature of chronic mastitis and chronic cystic mastitis and offer an entirely different and new classification. This is based upon conditions noted in the breasts as mastitis which are more physiological than pathological; and those noted with cysts and papillomas that are distinctly pathological. The physiological type of breast they call mazoplasia, the condition being found in some form in all breasts up to the menopause. The early pathological state of the breast they term cystiphorous desquamative epithelial hyperplasia, which in the literature in the past has been known as senile parenchymatous hypertrophy (Bloodgood), Schimmelbush's disease, Reclus disease, and chronic cystic mastitis. This condition begins in the later 20 years of life and there is found either a small cyst or numerous small and large cysts. As the cysts become older, during the late 30 or 40 years of life, there can occur certain neoplastic changes without any clinical indications or symptoms noticed by the patient. Possibly the first unusual symptom occurring in this stage is a discharge of serum or blood from the nipple with the appearance of an increase in pain. This would be indicative of a single or multiple benign papilloma or a carcinoma. The supervention of the neoplastic change is not a necessary consequence, and the cystic condition may remain in the pure state. These authors state that a carcinoma may arise in the apparently single cystic lesion, in the generalized multicystic state, and in those localized to a single duct, its branches, or acini. Malignancy appears about 25 to 30 years after the incidence of the cystiphorous stage and develops in small cysts in 20 per cent of all cases of carcinoma of the breasts. They further state that a carcinoma may originate in the ducts of the nipple which presents characteristic findings of a small, not very malignant tumor, giving a spontaneous discharge of blood from the nipple with early ulceration. This tumor most often arises from the duct epithelium and, according to Muri, is the cause of Paget's disease. When the tumor is immediately beneath the nipple, there may be an early fixation and retraction of the nipple and blood may be the very first symptom.

A discharge from the nipple may occur in the newborn. It is of a milky character and is associated with bilateral hypertrophy of the breasts.

[1] Read before the Western Surgical Association, St. Louis, Missouri, December 7, 1934

Fig. 1. Dilated duct with retained secretion. Periductal round cell infiltration. Dilation of ducts was noted in both peripheral and terminal ducts.

Fig. 2. Dilated duct with areas of single layer, other areas showing epithelium several layers thick. No papillomas were found in numerous sections.

It very shortly disappears by cleansing the nipples and the application of protective dressings. It may be complicated by infections, abscesses, or erysipelas with ultimate death.

In adults, a discharge from the nipple may be serous, thick greenish, whitish, or of a bloody character.

A serous discharge is usually unilateral and may occur with infection of the ducts of the nipple. Its presence should be a cause of anxiety, particularly when a tumor is discovered in the same breast, as such a discharge often accompanies malignancy. A patient with this type of discharge and no other malignant findings should remain under constant observation until the diagnosis of malignancy can be definitely eliminated.

A discharge of a thick, greenish material from the nipple occurs in young married women who have borne children. It usually is bilateral, is not spontaneous, and is caused by the retention of secretions in dilated ducts following lactation.

A whitish discharge simulating milk is associated with the more or less rare tumor, galactocele. This is a painless, circumscribed, fluctuating, doughy tumor occurring with lactation which cannot be very easily confused with any other pathological condition in the breast. The discharge may be white, due to pus, or in rare cases, to masses of desquamative epithelial cells.

Discharge of blood from the nipple may be traumatic, is easily recognized by the history and on examination there is evidence of ecchymosis. In determining the extent of the injury to the breast, assistance may be possibly obtained by transillumination. Should a hematoma be present, such an examination reveals a characteristic opacity, irregular in outline, more dense in areas than in others with very irregular edges which gradually fade at their margins. These findings may persist for as long a period as 3 months. By comparison, the shadow cast by a carcinoma is usually much lighter, fairly homogeneous, with more regularity of its edges; if cancer is suspicioned, further investigation should be made.

Papillomas are probably the most frequent causes of bleeding from the nipple, occurring usually in the late 30 or early 40 years of life. The principal etiological factor in the development of papillomas is conceded to be some form of irritation, either stagnation of the contents of ducts following lactation, stagnation of desquamated epithelial cells, micro-organisms, or possibly trauma. Papillomas of the breast can disappear as any wart, and spontaneous cures have been

Fig. 3. Dilated duct to left is filled with blood. Dilated duct to right shows broad base, sclerosing papilloma.

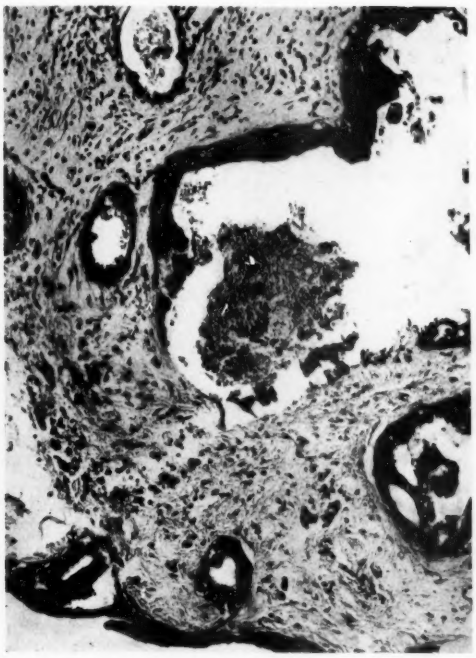

Fig. 4. Dilatation of ducts and epithelial hyperplasia of ducts but no extension beyond normal confines. The epithelial hyperplasia and hyperchromatism bespeak early malignancy.

reported. It is very doubtful, however, if the papillomas actually disappear. It is more likely they become quiescent with an absence of discharge of blood from the nipple and all other symptoms until possibly years later when symptoms again begin to appear.

Another breast lesion producing a discharge of blood from the nipple is Bloodgood's "Varicocele Tumor of the Breast." This mass is found to be single, multiple, or to be present with other pathological conditions. Patients having this condition are at the menopause or have passed it. The tumor is characterized by a doughy and worm-like feeling, is painful and tender. The discharge from the nipple is grumous, brownish material varying in color. When this condition can be recognized clinically, an operation is not indicated.

The early recognition of Paget's disease and the radical removal of the affected breast affords greater success in the treatment of this condition. Although the pathological process of Paget's disease is slow, seldom infiltrates and rarely metastasises, a carcinoma very frequently exists in the underlying breast without any of its usual clinical symptoms or findings. Hertzler has found that cancer of the breast follows the onset of Paget's disease within 2 years and usually within 1 year.

A thin, clear, serous, straw colored fluid occurs only in the cystic forms of sarcoma. According to Gross, it is present in 10 per cent of the cases of sarcoma. The large size of the tumor, its circumscription, mobility, lobulation, elasticity, and the history of its rapid growth serve to differentiate it from carcinoma and, as a rule, from pure adenofibroma. In this pathological condition, there is no metastasis to glands, no adherence to the skin, and no retraction of the nipple.

A spontaneous and intermittent discharge of blood similar to that seen with a papilloma may be produced by a duct carcinoma or a carcinoma that arises from terminal ducts and acini combined. The discharge may occur at intervals of weeks, months, or years and its character is no indication of the type of the disease present, malignant or benign. Pain is always present in these pathological conditions, varying in extent from a mere discomfort in the breast to the severe pain of distended ducts.

The diagnosis in the early stages of both benign and malignant conditions is often hindered by the absence of a demonstrable tumor. In a case of

Fig. 5. Large compound papilloma which was found in dilated duct. It measured about five-tenths centimeter in diameter. There was no evidence of malignancy.

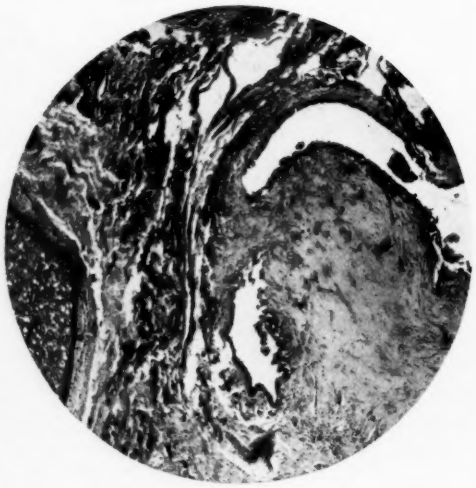

Fig. 6. Large compound papilloma extending into dilated duct with sclerosis of portion of papilloma. There was no evidence of malignancy.

bleeding from the nipple with no palpable tumor present in the breast, the roentgenographic findings being negative, one can conclude that a papilloma is present in one of the ducts. The exact location of a papilloma can frequently be determined by locating a tender point and exerting pressure on it, when blood is expressed from the nipple. When the papilloma is palpable, it appears to be very elastic, elongated, is freely movable and lacks the hardness of a carcinoma.

The variance of opinion regarding the significance of bleeding or discharge from the nipple and its bearing on treatment lead to a review of the records of all patients with breast conditions that had been treated at Research Hospital the past several years. Our study revealed only 10 cases that had at some time during their clinical course noticed bleeding or discharge from the nipple. These patients were operated upon by various members of the staff, and the specimens removed at operation were examined and reported upon by the hospital pathologist, Dr. F. C. Narr.

CASE 1. Hospital No. 83474. Mrs. V. W., aged 44 years; 3 pregnancies. Discharge had been noticed from the right nipple for 2 weeks, the discharge at first resembling milk, later becoming blood tinged. The nipple was sore and under it the patient could feel a small mass which was not tender. A simple amputation of the breast was done. Pathological examination revealed all of the ducts to be markedly dilated and filled with a light green, soft, putty-like matter. About them there was a localized connective tissue proliferation but no cyst cavities. Sections showed great distention of peripheral and terminal ducts with retained secretions but no neoplasia. Round cell infiltration about some ducts and acini (Fig. 1).

This case represents the typical pathological changes of chronic cystic mastitis or which Cheatle classifies as cystiphorous desquamative epithelial hyperplasia.

CASE 2. Hospital No. 71473. Mrs. E. H., aged 37 years, with 1 child aged 6. A lump had been noticed in the right breast for 8 months, a neuritis in the right shoulder and arm for 1 year, and a "purulent" discharge from the nipple for 5 weeks. She had been treated with diathermy, hot applications, alcohol dressings, and balsam of Peru. Examination revealed a freely movable mass the size of a lemon, fixed at the nipple. Cord-like strands could be felt radiating outward from the tumor mass. At operation a mastectomy was done. There was noted considerable fluid of a serous to "purulent" character escaping into the incision from the glandular structure. Numerous cut sections of the breast revealed multiple blue, dome-like cysts. Microscopically, there is some sclerosis of the fibrous tissue stroma of the breast and several cysts, varying considerably in size, lined with cuboidal epithelium which in some areas are several layers thick. No evidence of malignancy was noted (Fig. 2).

CASE 3. Hospital No. 82213. Mrs. C. H., aged 32 years; married 11 years, and has 3 children. Patient complained of marked intermittent tenderness in the left breast beginning about 9 months previously. Four months before admission she noticed a large lump in the dependent portion which disappeared in 1 week by the use of a sling, a support, and the application of heat. Three weeks before admission she noticed a bloody discharge from the nipple and since then there has been an occasional drop from it from day to day. Examination revealed a slight sanguineous discharge

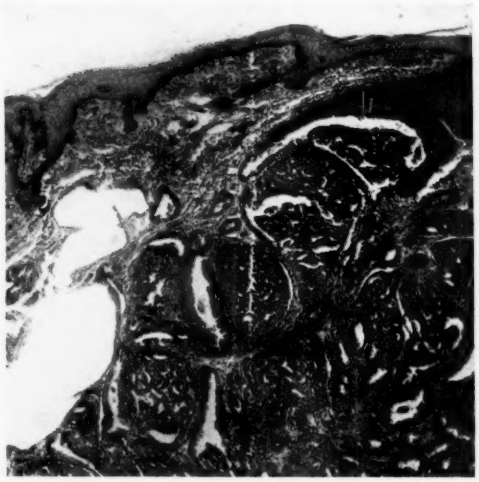

Fig. 7. Above skin surface. Terminal ducts with epithelial hyperplasia. Other sections showed ductal papillomas and carcinomatous invasion of breast stroma.

Fig. 8. Above dilated duct with papilloma and secretion. Lower center groups of epithelial cells (carcinoma).

from the left nipple, no palpable masses, no tenderness. Roentgen examination revealed signs of a cystiphorous desquamative epithelial hyperplasia which, in the absence of a mass and bleeding from the nipple, permitted a presumptive diagnosis of papillomata to be made. Operation consisted of simple amputation of the breast. Multiple sections through the specimen showed an increase in the fibrous tissue of the breast with a slight brown and red discolored area underlying the nipple, measuring 3 centimeters in diameter, a plexus of dilated blood vessels. Microscopic sections of breast showed small adenomata. There is dilatation of the ducts with blood in some and cellular débris in others. Occasionally one sees fibrous projections into lumina of ducts covered with epithelium, about which there is fibrosis and round cell infiltration (Fig. 3).

Case 4. Hospital No. 79414. Mrs. M. V., aged 56 years, has 2 children, aged 35 and 26 years. She gives a history of some pain and slight "purulent" discharge from the right nipple of 10 weeks' duration. Examination revealed a tender mass in the right breast with a "purulent" drainage from the nipple which was slightly ulcerated. X-ray examination revealed the findings to be those of a cystic condition with degeneration that was probably malignant. At operation a radical amputation of the breast was done because of a pre-operative diagnosis of Paget's disease. Grossly, the specimen presented a hard, irregular mass measuring on section 2.5 by 2 by 5 centimeters. There was considerable cystic degeneration, none of the cysts measuring more than 1.5 centimeters in diameter. The tumor was not encapsulated and from its periphery radiated pinkish white strands of rather dense tissue, into the surrounding stroma, suggestive of malignancy. Microscopic sections show dilatation of ducts with epithelial hyperplasia and the formation of small papillomas projecting into the lumen. There is no invasion of the stroma. In other areas there is epithelial hyperplasia with a filling of the lumen of the duct and with definite periductal round cell infiltration. This case is definitely precancerous as evidenced by the rapidly growing epithelial cells (Fig. 4).

Case 5. Hospital No. 87992. Mrs. L. P., aged 33 years; single. Patient had had a swelling in her left breast 7 months previously which disappeared following a serous discharge from the nipple. This occurred on two occasions. Four or five weeks before admission, the discharge became sanguineous. There was some soreness and a pain which radiated down the medial surface of the left arm to the wrist. Examination revealed a brownish pigmentation of the skin of the breast, no asymmetry. A small mass was palpable in the upper inner quadrant and when pressure was made upon this mass, a brownish fluid escaped from the nipple. No adenopathy, no retraction of the nipple, no tenderness were noted. Operation consisted of an excision of the tumor with a zone of the breast tissue. The specimen revealed several ducts distended with a brownish, thick fluid matter, suggesting changed blood. In one of these dilated ducts a glistening, grayish, soft papillary tumor hung from the wall, measuring about 0.5 centimeter in diameter. Sections show a papilloma of duct but no evidence of malignancy (Fig. 5).

Case 6. Hospital No. 80939. Mrs. M. H., aged 50 years, has 2 children; three miscarriages. She had a bloody ooze from the right nipple of 1½ months' duration. A few drops of blood in 24 hours occurred at first, later to change in consistency to a serous, thin fluid. For 1 or 2 years she had noticed some itching of the nipple but no pain until 2 days before admission. Examination revealed no lumps in the breast. At operation several pieces of tissue were excised, showing it to be very fibrous and of a firm consistency. No gross evidence of inflammation or tumor formation could be found in the specimens, although one small, thin area of connective tissue was thought to be a cyst wall. Microscopically, in one of the numerous sections is seen a distended duct with a projection of granular tissue, some of the epithelial cells of which appear to invade, but they do not impress us as being of active growth. The growth was believed to be benign (Fig. 6).

Fig. 9. Terminal duct showing marked epithelial hyperplasia. Other areas showed definite malignancy. Clinically, the case was one of Paget's disease.

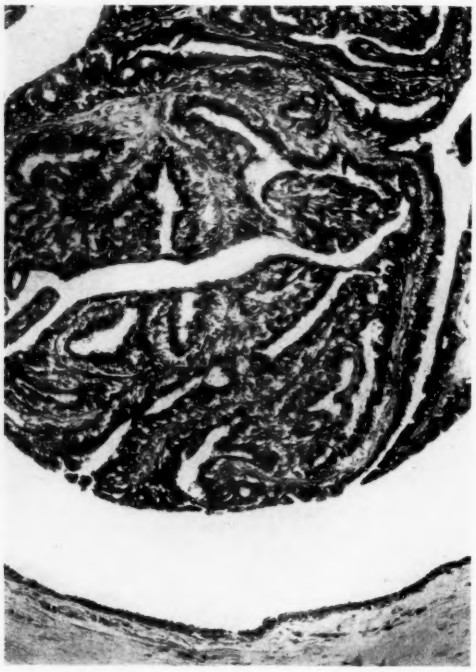

Fig. 10. Papilloma of duct, the epithelium of papilloma large and hyperchromatic. This breast in other sections presented malignancy.

CASE 7. Hospital No. 91250. Mrs. M. M., age 68 years, had no children, menopause occurred 12 years ago, patient stated that at that time she had some bleeding from the right nipple then she noticed nothing until 6 months ago when there appeared a lump in the breast. This increased in size but softened after some bleeding from the nipple. A hard, small, slightly tender mass was felt to the left and below the right nipple. Operation consisted of excision of a zone of breast tissue with the nipple. On sectioning the specimen, there was found underlying the nipple a rounded, solid, grayish granular mass located within or in close proximity to the ducts. The ducts showed several areas of cystic dilatation and contained dark red blood or soft grayish caseous matter. Microscopic sections showed a cystic dilatation with epithelial hyperplasia of ducts with a low grade papilloma formation. In one area at least there was thought to be an extension beyond normal confines. The pathologist considered this to be a ductal carcinoma of low grade malignancy (Fig. 7). A frozen section in the operating room was diagnosed papilloma with no evidence of malignancy. A report from the laboratory of the complete routine examination of the tumor disclosed the extension of the tumor beyond its normal confines. A radical operation for the removal of the breast was not done but the patient shows no evidence of recurrence at the present time.

This case exemplifies the great danger one occasionally encounters in the surgery of breast tumors.

CASE 8. Hospital No. 70840. E. F., aged 50 years, single, nurse. In June, 1927, she experienced an insect bite on the nipple of her right breast which was followed by redness, swelling, and tenderness, which disappeared in a few days. A few months later there occurred a discharge of some serous fluid from this nipple. Her physician advised cleansing of the nipple with alcohol. Four months before admission there was a discharge of a considerable amount of blood. Two weeks before admission the breast was accidentally struck with resulting swelling, tenderness, and considerable hemorrhage from the nipple. Examination showed a large, flattened lump which was not very tender and which seemed to be attached to the pectoral fascia. The nipple was not inverted and its pit was filled with a serosanguineous fluid. The skin over the upper pole of the tumor seemed to be attached to it. The right axilla presented no palpable glands. A radical amputation of the right breast was done at operation. The specimen showed a mass the size of a hen's egg just below the nipple, which was hard, fixed, and on cross section presented a very dense appearance of malignancy. On microscopic examination the breast appears to have been the seat of a mastitis. There are old sclerotic areas present with cyst-like cavities and papillary projections. In addition, there is a very definite malignancy outside of the area just described. The malignant cells are seen to produce small cords but nests and glands are also produced. There is a very definite invasion of the breast fat (Fig. 8). This case presents the interesting feature of having a papilloma present as well as a carcinoma in another area of the breast.

CASE 9. Hospital No. 89213. G. S., aged 55 years, had 1 child; menopause, 2 years ago. A lump in the right breast was first noticed 3 years ago. This was removed by a plaster. One year later another lump appeared and during the past 3 months it has increased rapidly in size. There was periodical bleeding from the nipple and it was raw at times. On examination there was found an eczematoid eruption

about the right nipple. A hard mass was present in the lower left quadrant of the right breast which was fixed to the muscles and skin. In the outer and upper quadrant there was another mass which was softer and was not fixed. A gland was palpable in the axilla. Operation consisted of a radical breast amputation. Pathologically the skin surface of the specimens showed two distinctive lesions. One was about an area which was considered to be the areola and nipple. The nipple was flattened out and appeared to be a continuation of the surrounding skin. It was differentiated, however, by a thin, shiny gray epithelium, somewhat suggestive of scar tissue. This area was surrounded by a series of pin-point sized bleeding points. The other lesion of the surface was a large contracted scar measuring 5 by 3 centimeters. The scar was adherent to a large, solid, hard granular mass which, on sectioning, proved to be a large, solid, hard, granular mass with definitely infiltrating bodies. On one edge of the mass was a cystic cavity 1.5 centimeters in diameter, filled with blood. On the tumor side of the cyst were papillary projections into the cyst cavity. The latter were apparently continuations of the tumor tissue. Sections through the nipple area revealed a marked prominence of the ductal structures. Three enlarged lymph nodes were found, one of which was solid, grayish, firm, and with glistening cut surfaces, suggesting malignancy. Microscopically, the sections showed skin inflammation under the epiderm but no typical Paget's cells. There was a suggestion of malignant cell invasion of the epiderm but this was not certain. The ducts of the nipple showed epithelial hyperplasia and extension beyond confines. Section of the tumor showed carcinoma of the ducts (Fig. 9). This case typically exemplifies the pathological picture of Paget's disease and its close association with a carcinoma of the ducts lying immediately beneath the nipple. The treatment of Paget's disease of the nipple should always be radical and the case treated as a malignancy from the beginning.

CASE 10. Hospital No. 82179. Mrs. E. P., aged 71 years, menopause at the age of 51, 8 children. Three years ago she noticed an inversion of her right nipple and about 6 months later a lump appeared in the right breast. Three weeks before admission to hospital she bruised her right breast which was followed by slight tenderness and a serous discharge from her right nipple. Examination revealed the nipple to be inverted and from it there was a serous discharge. A mass was present in the medial upper quadrant which was freely movable and not fixed to the skin. Some tenderness was noted on pressure. A simple breast amputation was performed; no frozen section was made and the axilla was not disturbed. The specimen had a retracted nipple in its center. A hard, rounded, somewhat nodular and not definitely circumscribed mass could be palpated within 1 centimeter from the nipple which had a slight attachment to the skin. Cross section revealed a mass 3.5 centimeters in diameter. Its cut surface showed a firm, pink and gray, granular friable tumor tissue with small hemorrhages and many areas of apparent necrosis at its center. The larger ducts contained much light yellowish green exudate. The microscopic pathology of many sections showed no evidence of malignancy. The stroma of the breast in these sections was translucent, did not appear to be as dense as hyalin. Other sections show undoubted malignancy. The condition is believed to be a cystoma which has undergone malignancy, the epithelial cells of the new-growth appearing at times to be spindle-shaped (Figs. 10 and 11).

The etiology of the bleeding from the nipple in this case is problematical but trauma was presumably responsible. The clinical features of the case were sufficient to warrant the surgeon making a pre-operative diagnosis of a possible malignancy. At operation, however, the tumor was not sectioned or a frozen section was not requested. The final diagnosis from the laboratory was received after the patient had left the hospital. Again, the importance of an immediate frozen section in the operating room cannot be stressed too strongly.

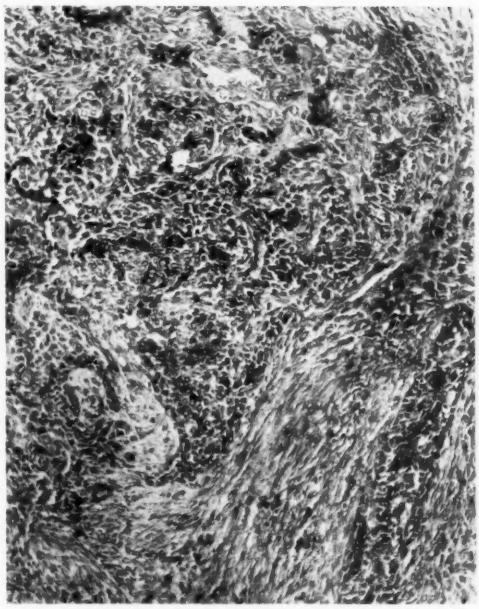

Fig. 11. Carcinoma in breast which presented the intraductal papilloma seen in Figure 10. Note duct filled with malignant epithelium in lower right corner.

SUMMARY

A careful study of the pathology of these ten cases reveals the interesting changes and gradations that occur in the breast from the very early cystiphorous desquamative epithelial hyperplasia state through to the neoplastic state of carcinoma as has been described by Cheatle and Cutler.

Cases 1 and 2 present the very early cystiphorous state and Case 4 with beginning neoplasia gives the history of there having been a so called purulent discharge from the nipple. The history of such a discharge is not described by Cheatle as being one of the early symptoms of this pathological state or of the early neoplasia state but he describes such a discharge as being associated with carcinoma. A discharge of blood may occur

in any of the stages as shown by this analysis even in Case 1, which is a very early cystiphorous lesion.

The 4 cases of malignancy in this group had a palpable tumor in the breast and serous or bloody discharge from the nipple. In Case 7 the discharge occurred 12 years previously and was followed by a carcinoma. These 4 cases exhibited pathological evidence of a malignant condition involving the ducts of the breast and one of these cases was associated with Paget's disease of the nipple.

Clinical examination revealed no tumors in the breasts of Cases 3 and 6 but in the 8 remaining cases, masses varying in size, consistency and sensitivity were noted.

The pathological examination of the specimens revealed papilloma to be single or multiple and when multiple they were found to be in separate ducts. Contrary to the opinion of some surgical pathologists, it was determined quite definitely that a papilloma may become malignant. Case 7 presented the history and physical findings of a simple ductal papilloma, was surgically treated as such, confirmed by microscopic frozen section but later there was found to be a carcinoma in the routine laboratory examination. Pathological examinations in Cases 9 and 10 show the neoplastic carcinoma with epithelial changes in the papillomas.

The analysis of these cases also confirms the often neglected surgical axiom that a lump in the breast should be removed as soon as it is noticed, regardless of the discharge from the nipple. Clinical signs of a carcinoma are necessary before a diagnosis of malignancy can be made and before these signs develop, the diagnosis of a palpable tumor can be made only microscopically. It is important therefore for patients having a tumor in the breast with a discharge from the nipple to be operated upon in a hospital with a competent pathologist at hand to make an immediate microscopical frozen section of the tumor. If found to be malignant, the surgeon should be prepared to perform immediately a complete radical amputation of the breast.

Inasmuch as papillomas are at times multiple and a carcinoma may lie obscured in another area of the breast, it is our opinion that the entire gland should be regarded with suspicion for immediate, as well as for future, pathological conditions. For this reason the surgical procedure of choice for all cases of bleeding or discharge from the nipple is a mastectomy. If a microscopical frozen section of a demonstrable tumor shows malignancy, the radical operation can then be done.

CONCLUSIONS

The study of these cases, although small in number, leads us to the following conclusions:

1. The pathological classification of benign and malignant tumors of the breast seems greatly facilitated by following the work of Cheatle and Cutler.

2. A discharge of blood from the nipple with no palpable tumor, in the absence of a history of trauma, is presumably caused by a papilloma of a duct or ducts of the breast.

3. A so called purulent discharge from the nipple is found to be associated with a cystiphorous desquamative epithelial hyperplasia as well as with carcinoma.

4. A discharge of serum or blood from the nipple with or without a palpable tumor is a surgical condition.

5. Mastectomy should be the surgical procedure of choice as papillomas may be multiple, may be present in more than one duct, and contrary to authorities, papillomas may become malignant.

6. An immediate microscopical frozen section of the tumor should be made in the operating room and, if this discloses a malignancy, an immediate radical operation should be performed.

7. Routine laboratory study of multiple microscopical sections of the breast is essential in discovering other pathological lesions.

8. A carcinoma of the breast may be hidden and obscured in one area of the breast while the clinical symptoms of a papilloma predominate in another part of the breast.

9. Paget's disease of the nipple can be considered as always being associated with carcinoma and demands a radical amputation of the breast.

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OBSTETRICAL ANALGESIA WITH PENTOBARBITAL SODIUM

THE LEUCOCYTE RESPONSE DURING THE PUERPERIUM

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RECENT clinical observations and laboratory studies point to a number of commonly used drugs which occasionally provoke neutropenia in susceptible individuals. The nature of the mechanism by which they depress the activity of the bone marrow is not clear, although the evidence seems to indicate that compounds containing the benzene ring are the chief offenders. Thus, amidopyrine and arsphenamine are among the more common drugs, the exhibition of which has been followed by neutropenia.

The administration of several popular proprietary mixtures of drugs which contain both barbiturates and coal tar products, such as allonal (allylisopropylbarbituric acid plus amidopyrine) and amytal compound (amytal plus amidopyrine) has been reported to have been followed by neutropenia. In the minds of many physicians this has brought under suspicion the derivatives of barbituric acid as a group.

With the exception of those which contain the benzene ring, (i.e. phenobarbital) there is no evidence that the derivatives of barbituric acid themselves cause neutropenia. Barbiturates have been used extensively and in relatively large doses over a period of nearly 4 years at the Boston Lying-in Hospital. There have been no deaths from neutropenia during this period, neither have there been any cases in which the diagnosis of neutropenia due to drug poisoning could be made. In this connection it is of interest to review in more detail a consecutive series of patients who have received such medication during labor.[2]

Our series consists of 3,592 delivered patients, 1,242 of whom received pentobarbital sodium [sodium ethyl (1-methyl-butyl) barbiturate] alone, and 2,350 of whom were given pentobarbital sodium in combination with scopolamine. The drugs were given in sufficient quantity to produce analgesia and amnesia during labor. When scopolamine was used, it was given by hypodermic injection. The pentobarbital sodium was given by mouth (rarely by rectum) in total dosage varying from 3 to 25½ grains. Few patients received less than 6 grains. The larger doses were divided, rarely more than 6 grains being given at once. The period over which the drug was given varied as the labor; in many, a single dose sufficed, in others, divided doses were given over a period of 48 or more hours.

There were no cases of typical agranulocytosis or neutropenia among the 3,592 patients. In the whole series there were 11 deaths, most of which were primarily obstetrical. We do not believe that neutropenia caused by pentobarbital was a factor in any of them. In order that this may be clear, every death in the group is briefly abstracted.

CASE 1. No. 1669. A multipara of 31 delivered normally after a short labor during which she received 9 grains of pentobarbital. The postpartum course was entirely afebrile and uneventful until she died suddenly on the eleventh day of massive pulmonary embolus.

CASE 2. No. 11175. A primipara of 25 at term suffered from severe rheumatic heart disease with both mitral and aortic lesions. She was admitted in labor on the edge of congestive failure. Three grains of pentobarbital were given

[2] Since this work was begun, a series of patients who had been given pentobarbital sodium during labor were reported by Hardwick and Randall (J. Am. M. Ass., 1934, 102: 158–160). The total number of patients reported was 59, only 5 of whom received more than 9 grains of the drug. Their finding—"Studies of the blood from this group of patients give no evidence of the production of a leukopenic condition with the amounts of pentobarbital-sodium administered" is entirely substantiated by our results.

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and she was delivered by low forceps when the cervix was fully dilated. There was more than the usual blood loss at delivery, and the patient was put back to bed in mild failure. She improved until the third day when suddenly she developed multiple pulmonary emboli from which she died, 24 hours later.

CASE 3. No. 7271. A primipara of 26 was delivered normally. During the course of labor she had received 9 grains of pentobarbital. On the fifth postpartum day, she developed signs of severe puerperal sepsis from which she died on the eleventh day. White blood cell counts on the fifth, sixth, and tenth days were respectively 27,000, 19,800, and 14,100.

CASE 4. No. 10714. A 40 year old primipara was delivered by cesarean section after a long test of labor, during which she received 4½ grains of pentobarbital. White blood cell counts on the third and fifth days were 24,600 and 14,100, respectively. She died unexpectedly on the eighth day of pulmonary embolus.

CASE 5. No. 6733. A quadripara of 30 with placenta previa was delivered by cesarean section. There was a profuse postpartum hemorrhage from which she died in spite of transfusion and hysterectomy. She had received 9 grains of pentobarbital.

CASE 6. No. 7779. Labor was induced during the thirty-eighth week in a multipara because of increasingly severe toxemia. The patient was delivered normally after a 3 hour labor during which she was given 6 grains of pentobarbital. There was a delayed postpartum hemorrhage from which she died, 9 hours after delivery, in spite of packing of the uterus and two transfusions.

CASE 7. No. 10662. A primipara of 22 was delivered by low forceps after a short labor during which she had received 6 grains of pentobarbital. She died on the twentieth postpartum day of bilateral caseous tuberculous pneumonia. Ten white blood cell counts in the first 18 days varied between 11,000 and 20,000.

CASE 8. No. 8412. A primipara of 24 was delivered by low forceps after a labor during which she received 9 grains of pentobarbital. Eight hours after delivery, she suddenly became cyanotic with rapid pulse and respirations. She died on the second postpartum day of pulmonary embolus.

CASE 9. No. 10084. A tripara of 24 was delivered by cesarean section after a test of labor during which she received 3 grains of pentobarbital. She died on the twenty-ninth postpartum day of sepsis. White blood cell counts on the sixth, eleventh, and sixteenth days were, respectively, 11,000, 28,000, and 10,000.

CASE 10. No. 11182. Because of severe neglected toxemia which had failed to respond to treatment, labor was induced in an obese primipara of 27 at term. After an 18 hour labor, during which she had received 9 grains of pentobarbital, she was delivered when the head had been on the perineum for 2 hours without progress. The baby was very large (11 pounds 3 ounces) and the delivery of the shoulders very difficult. The patient died 4 hours postpartum of shock and hemorrhage in spite of transfusion.

CASE 11. No. 61054. A primipara at term was delivered by transverse cervical cesarean section after a long test of labor, the latter part of which was febrile. During labor, she had been given 18 grains of pentobarbital. The patient developed peritonitis and bronchopneumonia. On the sixth day the wound broke down and she eviscerated her intestines. She died on the seventh postpartum day, 24 hours after secondary closure of the wound. White blood cell counts were as follows: 2 days following medication, 24,600; third day, 13,600, 11,600; sixth day, 3,700 and 1,800; and on the day of death, 5,150. Differentials showed polymorphonuclears in excess of 64 per cent, except for the count of 3,700 when they were 50 per cent.

The last case is the only one in the whole series in which the white blood cell count was low. We do not believe, for several reasons, that the administration of pentobarbital had anything to do with the low white count in this case. In the first place, the severity of the infection in itself would adequately explain the low count. Besides, there was a marked initial leucocytosis 48 hours after administration of the drug, which is not characteristic of the neutropenia which follows the administration of drugs. Finally, the count was never extremely low, and the percentage of neutrophils was never below 50.

Since in the course of routine postpartum care, white blood cell counts were done only upon patients with complications in the puerperium in whom there were clinical indications, it is impossible to state that there were no instances in which the white cell count was abnormally low. However, one can state that there were no patients with clinical symptoms referable to neutropenia.

With the possibility in mind of low white counts following use of pentobarbital in patients without symptoms, a series of white blood cell counts were made during the puerperium upon a group of patients who had received the larger doses of the drug during labor. The counts were done when possible on the second and fifth days following delivery, and once or twice thereafter. This interval following administration of the drug would seem to be the most likely one in which to find neutropenia if the medication had caused it. In known cases of idiosyncrasy to antipyretics, such as amidopyrene, the symptoms have usually followed the use of the drug within 1 or 2 days. Neutropenia usually reaches its maximum within 5 to 6 days. In addition, the bulk of experimental evidence would tend to place the life of the mature circulating granulocyte at 3 to 5 days. Thus, if exhibition of pentobarbital had inhibited the bone marrow from releasing granulocytes into the circulation, the diminution in their number in the circulating blood would already be marked by the second postpartum day, and extreme by the fifth day. The subsequent counts were made to rule out a rather unlikely delayed effect. The results are presented in Table I.

With the exception of the one case of fatal peritonitis and bronchopneumonia already presented in detail, and in which the low white count was undoubtedly the result of an overwhelming infection, there were no abnormally low white counts. It is perhaps of interest that the average

TABLE I.—WHITE BLOOD CELL COUNTS IN PUERPERIUM FOLLOWING THE ADMINISTRATION OF PENTOBARBITAL DURING LABOR

Dose of pentobarbital	Number of cases	First 3 days			4th to 6th days			7th to 14th days		
		Maximum	Minimum	Average	Maximum	Minimum	Average	Maximum	Minimum	Average
7½–9 grains	16	25250	9250	15900	14950	5450	11500	16450	6200	9250
10½–12 grains	13	23850	11300	16650	20950	6550	11800	18350	6350	10900
13½–25½ grains	8	29600	10300	18800	24000	3650	13500	17700	1800*	10100

*Differentials were made in all cases. The percentages of neutrophils were high or normal in all cases except for this one, in which the polymorphonuclears were never less than 50 per cent. This case is reported in detail elsewhere in the paper.

white count increased as the dose of pentobarbital increased. This may be explained in that the patients who received the larger doses had longer, and, on the whole, more difficult labors, following which one would expect more reaction.

SUMMARY

A series of 3,592 cases is presented in which relatively large doses of pentobarbital were given during labor. None of them developed clinical symptoms of neutropenia. Repeated white blood cell counts during the puerperium in a series of patients who had received the larger doses of pentobarbital revealed that the drug was not followed by lowered white count, and, indeed, that it did not interfere with the normal leucocytosis of the puerperium. Finally, there would appear to be no ground for fear that pentobarbital is followed by neutropenia when given in adequate therapeutic dosage during labor.

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OCTOBER, 1935

CHOLECYSTECTOMY WITHOUT DRAINAGE

WIDESPREAD interest in cholecystectomy without drainage has introduced new problems in the field of biliary surgery. Omission of the drain after simple cholecystectomy permits a smooth and rapid convalescence, less foreign body reaction with its consequent adhesions, decreased likelihood of dehiscence of the abdominal wound, and absence of an irritant drain rubbing on ligatured bile ducts and blood vessels, inviting late leakage.

Unfortunately, the universal desire to try new methods results in a backward swing of the pendulum. In the hope that the good results of cholecystectomy without drainage may not be lost in the reverberations against the procedure, a recapitulation of the indications and technique may be timely. The success of the method is granted, but a few failures have evoked a storm of criticism; the latter have been due, in so far as I am able to interpret them, to injudicious and inexperienced efforts in this new field.

It would seem that the indications for omitting the drain in cholecystectomy should be evident. Recent or old perforations of the gall bladder with resultant abscess formation, acute cholecystitis, and adherent omentum or viscera which must be freed from the gall bladder before the latter can be removed, warrant drainage in almost every instance. Injected, edematous tissue invites drainage, lest our judgment of the degree of subsiding inflammation be underestimated. Spillage of gall-bladder content, however minor, and all instances of surgical exploration of a bile duct, demand provision for drainage. There should be no doubt of drainage in all the instances cited, but if doubt persists, the safe dictum is, "when in doubt, drain."

Technically, cholecystectomy without drainage is on a par with thyroidectomy without drainage. When Wallace I. Terry promulgated thyroidectomy without drainage, he changed the technical details of the operation. The same principles advocated by Dr. Terry must be adhered to if we are to omit the drain when performing cholecystectomy. In only certain cases may operation be done without drainage and certain features of the technical detail must be assiduously observed. The major requisite is a dry operative field. All severed blood vessels and bile ducts must be securely ligated; the smaller sizes of ligature material should be used; mass ligation of tissue is contra-indicated and approximation of tissue without tension is pre-requisite. Raw surfaces may not be left exposed in the liver bed and meticulous attention to small hematomata is necessary.

When subserous cholecystectomy is performed after the injection of salt solution under the serosa of the gall-bladder wall, the

edema thus produced permits easy enucleation of the gall bladder, but it also may result in late oozing of blood or bile from the small vessels compressed by the edema. When the edema is absorbed, the vessels open and drain. Fortunately, the vessels are small and do not drain long, but they may ooze sufficiently to militate against the success of non-drainage. If no drain is used, these vessels must be ligated even though they do not bleed during the operative period. The same holds true for the small bile ducts which empty into the side of the gall bladder from the liver bed. As these anomalous structures are present in nearly 6 per cent of all persons, they offer a danger of considerable magnitude. Of course other anomalous biliary ducts must be searched for, but though they occur in at least 10 per cent of bodies they are not so frequently overlooked, for they are of larger caliber. Anomalous blood vessels are even more numerous, yet are rarely even searched for. If any bile ducts are encountered that seem to be anomalous, they should be exposed from their origin in the liver to their termination in the gall bladder, bile duct or duodenum. The cystic duct should be exposed from the gall bladder to the common hepatic duct by bluntly dissecting the peritoneum and fat from it, for unless it is amply exposed, anomalies of the ductal and vascular tree may be traumatized without recognition, to become a source of late leakage. Gentle handling of tissue and atraumatic technique are vital to the success of cholecystectomy without drainage. The hurried operation, strenuously performed, is no procedure to be followed by omission of the drain.

The anesthetic is important, for when the patient must be heavily drugged in preparation for the anesthesia, and in cases in which ether is used as the anesthetic agent, bile flow from the liver is decreased. Then the smaller biliary ducts do not drain when severed and therefore they are not secured by ligature. Local infiltration, spinal and gas anesthesias do not interfere with this function of the liver.

In spite of the limitations thus placed upon cholecystectomy without drainage, it has been possible to close the abdomen without drainage in 82 per cent of 241 simple cholecystectomies for gall stones. In only one instance has omission of the drain been regretted, yet in 2 cases drains have failed to function. In both of the latter, the drain itself became walled-off, and probably induced the bile lake which surrounded the terminal tip of the drain.

Cholecystectomy without drainage is a new field in biliary surgery and promises to be a satisfactory procedure, but it cannot be done successfully unless our technique is altered and of course, unless our selection of cases is judicious. STANLEY H. MENTZER.

DIAGNOSTIC STUDY OF OBSCURE ABDOMINAL COMPLAINTS

DESPITE all attempts at accuracy, diagnosis in obscure abdominal complaints is still inexact and far removed from the goal that medical men are seeking. While the art of administering treatment, both medical and surgical, can gradually be acquired by the individual, the making of diagnoses in obscure abdominal conditions usually requires co-operative effort; and while the treatment of disease makes a strong appeal to the more material side of the physician, the making of diagnoses seems to appeal more to the spiritual and intellectual side. Consequently, the making of diagnoses usually presents more difficulties than does therapy.

Vague abdominal complaints which persist present a large field for diagnostic study and are frequently slighted by the physician until

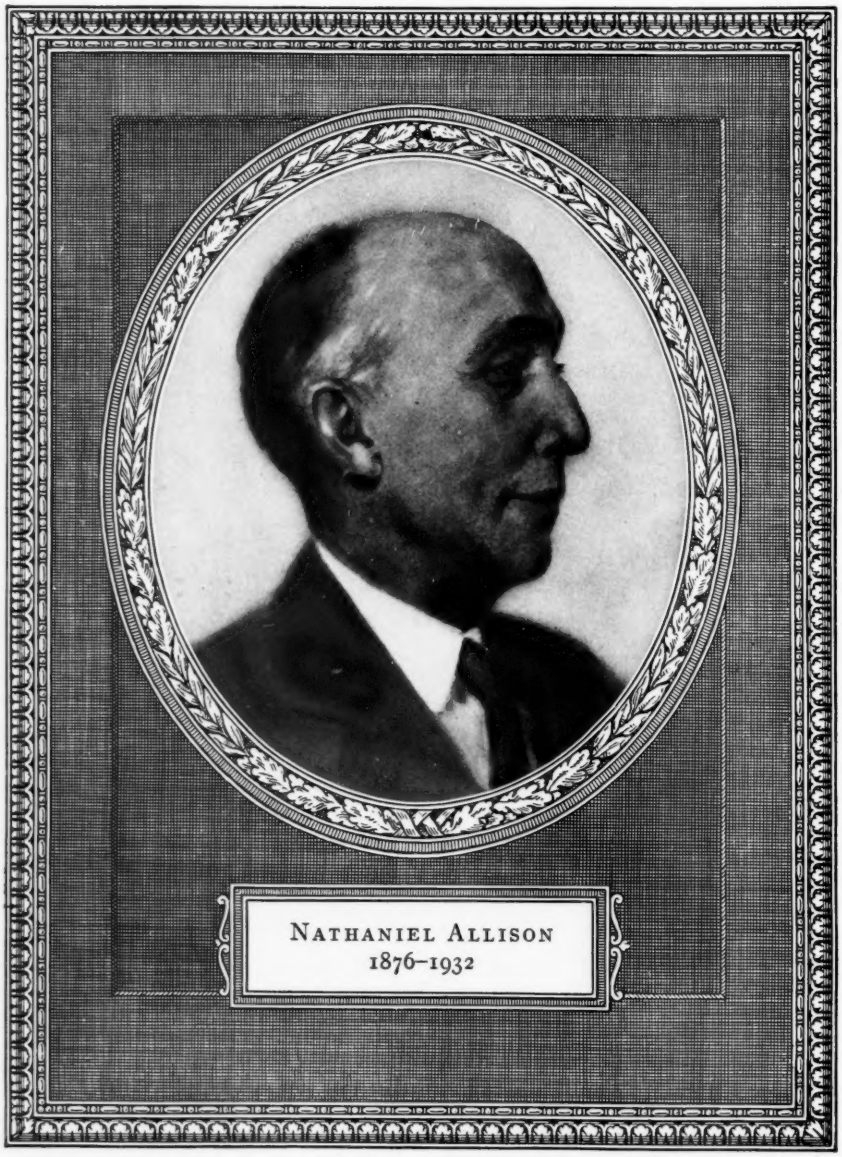
the clinical picture becomes sufficiently clarified to clinch the diagnosis. Often this is late in the progress of the disease. In other cases exploratory operations, which are more or less useless, are performed to clarify the situation.

With all the recent advances in roentgenography, many of these cases, if adequately studied, can be elucidated. Unfortunately, even with all these modern aids, in many cases diagnosis still remains obscure. The technique of studying these patients is still expensive, but the method of approach to these obscure conditions seems definitely crystallized since we can portray so much on the X-ray film. We have adopted a definite routine in these patients and this routine visualizes the whole abdomen—the urinary tract, the biliary tract, and the gastro-intestinal tract, and as well the subarachnoid space, if there is any suggestion of its involvement.

First a flat picture of the abdomen is made and then an excretory urogram. A series of pictures of the gall bladder is followed by a series of the gastro-intestinal tract and of the colon after a barium enema. In this way the three important tubular systems within the abdomen are studied with almost scientific accuracy, and as most complaints arise from disturbances in these parts, the patient's condition or complaints may thus be clarified with some certainty.

In cases in which the roentgenographic studies indicate that the complaints are not connected with these tubular organs and in which there is adequate suspicion that the abdominal pains are referred, a lipiodol subarachnoid injection is indicated for diagnosis and localization of a possible spinal cord neoplasm. This procedure may clarify a previously perplexing clinical picture. All in all, adequate and complete use of the available diagnostic methods in abdominal complaints is not made at the present time. It is definitely our duty to study all obscure cases completely in the manner here again outlined.

EDWIN BEER.



NATHANIEL ALLISON
1876–1932

MASTER SURGEONS OF AMERICA

NATHANIEL ALLISON

NO one member of the medical profession in this country has had more influence on the development of modern orthopedic institutions than Nathaniel Allison. As professor of orthopedic surgery in three of our leading medical schools at various intervals during the last 15 years, and as chief of the orthopedic services which these schools controlled, his influence was widespread and of the utmost importance in their development. Those who were fortunate enough to work under him experienced a stimulus rarely felt. Always alert to the newer developments in surgery of the bones and joints, and interested in investigative work, he made all who came in contact with him feel the urge to improve the whole field of orthopedic surgery. His own investigations were most valuable, but only with the realization of the volume of work produced by his associates, often at his suggestion and always with his fullest co-operation, can the scope of his influence be appreciated.

Dr. Allison's influence was not confined alone to investigative work in surgery of the bones and joints; he excelled also in organization. In the problems of hospital organization, of work by committees, whether of a medico-political trend or of the most scientific import, his ability was outstanding. Scientific meetings, large or small, were led by his quiet but firm influence. He was able to bring together a conglomeration of divergent viewpoints, and thus develop order out of chaos.

Dr. Allison was born in St. Louis. His father was the son of Dr. Nathaniel Allison of Mexico, Missouri, and his grandfather's influence led him to enter the practice of medicine. His early education was obtained in St. Louis. This was followed by a period of study in the United States Military Academy in West Point. He entered Harvard in 1894, and graduated in medicine there in 1901. After an interneship at the Boston Children's Hospital and a period of service with Dr. Edward Bradford in Boston he returned to St. Louis in 1904, where he built up his reputation as an orthopedic surgeon of international repute. He was married to Marion Aldrich of Chicago in 1909.

Dr. Allison's earlier work in St. Louis led to the development of the orthopedic service at the Barnes Hospital and the teaching of that branch in the Medical School of Washington University. In 1915 he was with the American Ambu-

lance Corps at Neuilly, France, and later with the American Expeditionary Forces he served in Base Hospital 21 at Rouen. In the autumn of 1917, when special consultant services were organized, he was second in charge of the work of organizing the care of fractures, and the work in the advanced areas was under his supervision. This was a task requiring constant vigilance, tact, and wisdom, and the efficiency with which it was carried out testifies to his skill and understanding. Returning from France he served for a time in Walter Reed Hospital as assistant director of the surgical service. He was awarded the Distinguished Service Medal and was finally discharged from the service as colonel, which rank he held in the Medical Reserve Corps until his death.

After Dr. Allison's discharge from the army he was sent to Rome as representative of the Medical Department at the Inter-Allied Congress of Surgery. Having accomplished this mission he returned to St. Louis and resumed his practice, and a professorship at Washington University, officiating as Dean of the Medical School. In 1923 he returned to Boston as chief of the orthopedic department of the Massachusetts General Hospital, and on the death of Dr. Robert W. Lovett he was made professor of orthopedic surgery in the Harvard Medical School. He also served as chief of the staff of the New England Peabody Home for Crippled Children, and in many other ways made his influence felt in the medical community of Boston.

In 1929, with the reorganization of the Medical Department of the University of Chicago and the Billings Memorial and Allied Hospitals, a chief of the Orthopedic Service was sought and Allison was asked to serve in this capacity, and again the result was one of the best organized and equipped orthopedic services in the country. While there, in 1930 he was stricken, and although he recovered and resumed his post for a period of many months in 1931, he never completely resumed his former activity.

His influence was wide, and many organizations not mentioned owed to him a great deal for his share in their development. His pleasing personality, and his ability to meet people of widely divergent viewpoints and bring them together when necessary, must be regarded as one of his outstanding characteristics. Those who worked with him always felt that he was governed only by a desire to see progress in orthopedic diagnosis and practice. Never without a plan to improve conditions when necessary, he was greatly in demand when all sorts of difficult tasks had to be performed. Although to many his work may not have seemed difficult, yet there must have been numerous times when the multiplicity of these tasks wore harder on him than was realized, thus helping to bring to an untimely end one of the most useful careers in modern orthopedic surgery.

RALPH K. GHORMLEY.

EARLY AMERICAN MEDICAL SCHOOLS

BAYLOR UNIVERSITY COLLEGE OF MEDICINE

CURTICE ROSSER, M.D., F.A.C.S., DALLAS, TEXAS
Professor of Proctology, Baylor University College of Medicine

BAYLOR University, the oldest university organized in Texas, was chartered in 1845 by the Republic of Texas during the Presidency of Anson Jones and for 90 years has occupied front rank in the educational affairs of the Southwest. The academic department has for many years been located in the centrally situated city of Waco, Texas; the various professional schools are in Dallas.

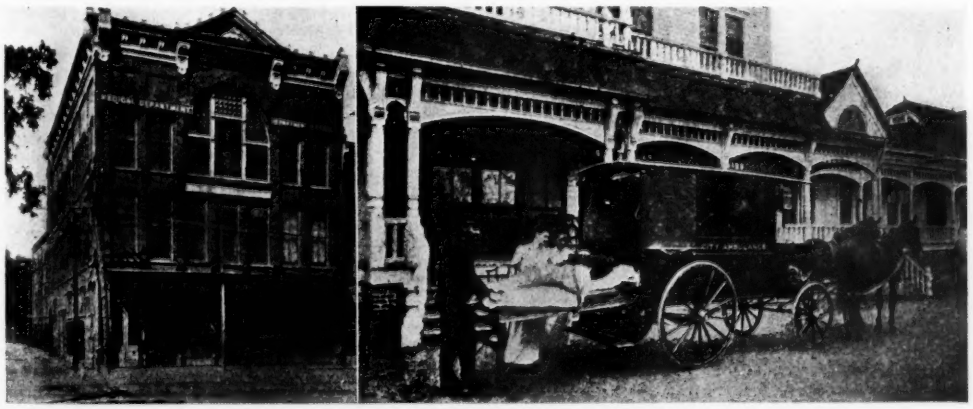
When, 35 years ago, a small group of physicians in North Texas determined that opportunity for medical education should be offered ambitious young men of this section, prevented by transportation difficulties and economic stress from attendance at apparently distant institutions, the establishment of a medical school in the then thinly settled and geographically isolated region was as surely a pioneer undertaking as had been similar ventures in eastern and middle western states a half century before.

After a long series of conferences, organization was effected in September, 1900, at a public meeting of interested civic and professional individuals.

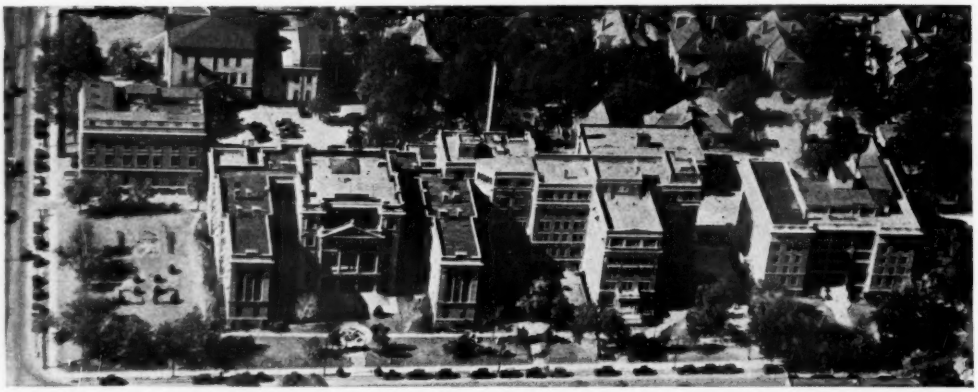
The first term of the new school, which was chartered by the State of Texas as the medical department of a projected but non-existent institution, the University of Dallas, opened on November 19, 1900, in a building which had formerly housed a Jewish synagogue.

To the astonishment of the new faculty, about 60 students registered for the courses, but whatever optimism may have been engendered by this apparent justification of the venture was soon dampened by a series of minor catastrophes.

A present perusal of the old minutes of the business meetings of the faculty during the organization's first year suggests to the reader the scenario of a melodrama with frequent interpolations from the book of Job. The equipment was meager, the teaching staff was small, and, although enthusiastic and earnest, its members with a few exceptions were unaccustomed to pedagogy. The majority of the local medical profession scoffed at the enterprise and freely predicted its failure. No state law permitting the legal use of cadavers for dissection and demonstration existed, and while the local authorities were sufficiently complaisant to render grave robbing unnecessary, the securing and transportation of anatomical material immediately became a problem. A well authenticated incident relates the midnight experience of one R. W. McFerran, a member of the first class, who volunteered to transport a body, unclaimed by friends or relatives, from the city morgue to the dissecting room with the tacit consent of those in charge. Mr. McFerran had only a horse and buggy for the purpose, and there-



The medical school building, left, and City Hospital, where ward rounds and clinics were first conducted, in 1903.



Baylor University of Medicine and Baylor University Hospital, 1935.

fore dressed the corpse carefully and placed it in proper sitting position beside him in the vehicle. Half way to the school building on turning a corner the body lurched and its hat blew off. A kindly policeman called out to the student "Your friend's lost his hat!" and as the officer ran behind the equipage with the head gear in his hand, the frightened embryo scientist plied the whip with vigor, shouted "he says you can have it" and, history records, reached medical sanctuary safely.

In December, 1900, three members of the faculty staged a revolution, took charge of the building by an overnight *coup d'état*, changed the name and organization of the school, and engaged a policeman to guard the premises in support of the new government. The remainder of the faculty were successful in securing an injunction against the junta forbidding interference with the proceedings of the medical department of the University of Dallas and a deputy sheriff dissolved the revolution. Fifteen diplomas were granted at the close of the first term, the recipients of which had obtained advanced standing from courses which were taken in other institutions.

In 1901, the minutes record, a permanent teaching staff of 17 was selected. In the allocation of professorships, surgery was assigned to Drs. Joe Becton, Sr., and B. E. Hadra; anatomy, to Dr. J. M. Inge; physiology, to Dr. A. C. Bell; pediatrics, to Dr. A. F. Beddoe; obstetrics, to Dr. J. H. Florence; gynecology and rectal diseases, to Dr. V. P. Armstrong; and gynecology, to Dr. J. E. Gilcreest. Dr. Elbert Dunlap, who is now head of the department of gynecology, was made professor of materia medica and genito-urinary diseases; Dr. Edward H. Cary, who later became dean, and who still holds the chair of ophthalmology and otolaryngology, was made professor of diseases of the eye, ear, nose and throat; Dr. Charles M. Rosser, one of the original founders group, who is now professor of clinical surgery, was made dean of the faculty.

Drs. Gilcreest, Becton, and Inge were well known and well trained Texas surgeons of the period who, living in neighboring communities, gave the infant institution helpful prestige and at considerable inconvenience lectured regularly to its classes. It is a matter of some personal interest to this recorder, in view of the fact that Baylor is now one of the twelve schools of the country including a complete section on proctology, that this earliest faculty listed instruction in the subject.

In 1902, the school building was destroyed by fire, and classes were conducted in the basement of the city hall until the conclusion of the term, when 19 graduates were awarded diplomas.

In 1903, the institution, in common with others throughout the United States, came to the crossroads of its destiny. For no other reason than that the plan was common and well understood the school had been organized as a stock company. The American Medical Association, in pronouncements issued in 1903, very correctly discouraged this type of organization and also insisted that university affiliation and teaching hospital connection were essential to those medical schools which were destined to survive.

Overtures were made to Baylor University looking toward affiliation and on June 29, 1903, an agreement was entered into whereby the stock company was dissolved and its properties conveyed to the university which assumed a protectorate over the school. In 1909, it became an integrate and coordinate part of Baylor University. Dissolution of the stock company and university affiliation satisfied two of the three criteria set up; the third requirement was met when, in 1909, the institution now known as Baylor University Hospital, which has been built on the grounds of the Good Samaritan Hospital, Dr. C. M. Rosser's private sanitarium, opened its doors and, operated jointly with the school, afforded an excellent "teaching hospital" connection.

The Rockefeller Foundation survey in 1908–1910 which disclosed widespread defects in the medical education of that time and resulted in a marked reduction in the number of existing medical schools, stimulated Baylor to necessary local corrections and additions, and in 1916 the Council on Medical Education of the American Medical Association awarded the institution the complete recognition which has been continuously maintained.

The faculty now number 144, 34 ranking as professors. The school hospital plant now represents a capital outlay of nearly two and one-half millions of dollars. Administration is under the direction of Dr. J. F. Kimball, vice-president of Baylor University in charge of professional schools; Dr. Walter H. Moursund, professor of bacteriology, has been dean of the faculty since 1923. The student body averages 360, the freshman class being limited to 100; degrees have been awarded 1,065 students, who now practice their profession creditably not only throughout the Southwest but in all parts of the United States.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE present volume of the *Surgical Pathology of the Peritoneum*[1] by Hertzler deals with the surgical pathology of the peritoneum and has 304 pages with 201 excellent illustrations by Tom Jones and Barlow. A comprehensive index is included and a short bibliography is given at the end of each chapter.

Professor Hertzler writes in a very clear and forceful style. It is apparent from this monograph that the author not only has interpreted a liberal amount of experimental work but has drawn freely on his large clinical experience. In reading the work, one is pleasingly impressed with the many practical applications and deductions.

From the viewpoint of a general surgeon, it was very thoughtful of the writer to restrict the discussion of the histopathology to a brief review of the important and characteristic changes. This conservative manner of presentation seems to reside almost entirely with writers of wide clinical experience. One has the impression that the author overemphasized the benignity of adhesions when he stated that undesirable clinical effects of permanent adhesions are negligible (p. 55). This seems somewhat incongruous in the face of the teaching that from 50 to 65 per cent of cases of acute intestinal obstruction are due to postoperative bands and adhesions.

The chapters on peritonitis are written exceedingly well and should be read by every young surgeon. Tumors of the peritoneum, mesentery, omentum, and retroperitoneal space are considered in one chapter. Still other chapters are devoted to diseases of the omentum, mesenteric thrombosis, and specific peritonitis.

This monograph is a distinctly worth while effort. R. W. McNEALY.

THE seventh edition[2] of Macleod's standard text of physiology is remarkable for the comprehensive revision which has been undertaken. The contributions of those assisting in the present edition have been skillfully welded into a harmonious whole, with little of the overlapping and repetition which is so frequently present in works produced by a group of collaborators. Many of the new concepts which have been developed in the 5 years which have elapsed since the previous edition are included without a material increase in the number of pages of this already large volume.

One of the chief merits of this text, applying equally to its use in the hands of students or for general reference, is the clarity of its presentation and the ease and pleasure with which it may be read. Most subjects are set forth in a detailed manner, in a style not ambiguous or likely to be found confusing. The 297 illustrations are for the most part clear and useful. A rearrangement of the contents, by which the treatment of the nervous system is placed last, introduces the student to those aspects of physiology which are perhaps most easily assimilated and allows of an orientation in the subject before introducing muscle and nerve which are often found more difficult. An excellent feature, which would bear adoption in other texts, is a rather comprehensive list of references which includes not only other texts and monographs but original papers in the various fields. From the American point of view, there is perhaps a disproportionate attention to English and continental literature while many significant American contributions are neglected.

While complete in many aspects, this edition, like those of previous years, entirely neglects certain phases of the subject. The term "Biochemistry" is now dropped from the title, and the aim of this work is to present physiology, with biochemistry "retained only so far as it is necessary to the elucidation of problems of animal function." The general subject matter covered, however, is much the same as in previous editions. Reproduction is still omitted; the liver is not given a section but is treated under various headings in the chapters on metabolism. A number of less important omissions detract from the completeness of the volume. The index is less complete than is desirable for one who uses the text primarily for reference.

To the medical student, perhaps the most serious lack is the failure to summarize the evidence concerning various phases of the subject and to draw specific conclusions. The volume is so large and the material in each section so voluminous, that while one can scarcely comment adversely on the absence of didactic presentation, brief recapitulations might bind the various chapters more closely and aid the student in obtaining a perspective of the science of physiology as a whole. A. C. IVY.

[1] SURGICAL PATHOLOGY OF THE PERITONEUM. By Arthur E. Hertzler, M.D., 1st ed. Philadelphia, Montreal & London: J. B. Lippincott Co., 1935.

[2] PHYSIOLOGY IN MODERN MEDICINE. J. J. R. Macleod, M.B., LL.D., D.Sc., F.R.C.P., F.R.S. 7th ed. St. Louis Mo.: The C. V. Mosby Co., 1935.

THE popularity of Sutton's well known work[1] is attested by the fact that this is the ninth edition. The author has adopted his son as a permanent literary associate and the work is now published under the joint authorship of Drs. Sutton and Sutton.

The book has always been an easy one to read as it is printed in large, legible type, on an excellent quality of paper. In this edition, 27 new conditions have been described and many others, including coccidioidal granuloma, tularemia, and the treatment of syphilis completely revised.

The illustrations are even more plentiful than they have been in the past, and in this edition there are 1310 illustrations and 11 colored plates. Many new photographs have been inserted to replace old ones and there are many photomicrographs.

The bibliography printed at the end of each subject has been added to and brought completely up to date. It has always been a feature of the work and is even more comprehensive than it has been in the past.

The only criticism that can be offered is that the book is growing so large that with further editions it may be necessary to publish it again in two volumes.

The authors are to be congratulated on the painstaking way the book has been compiled and I heartily recommend it to all practitioners who are interested in the subject of dermatology.

EDWARD A. OLIVER.

IN the seventh edition of *Physical Diagnosis*[2] by Elmer and Rose, the section on electrocardiography, silicosis, and diagnostic methods has been added to and revised. The first 543 pages are devoted to the physical examination of the entire normal body. Each of the standard diagnostic procedures is covered completely before the next one is considered. For example, the much neglected art of inspection is well described; the beginner completes his technique of inspection of the body as a whole, the head, neck, chest, abdomen, and extremities before proceeding with palpation, percussion, and auscultation, which sections are covered in a similar manner. This plan is especially suitable for the student who is a beginner in the study of physical diagnosis.

The second part of the book (340 pages) is devoted to diseases of the respiratory and circulatory organs. The section on electrocardiography and abnormalities of the heart beat is well written and contains numerous tracings. The chapter on "Radiology" in physical diagnosis is an essential part of any modern text on this subject. The text is abundantly illustrated and is recommended to both student and practitioner.

CLARENCE J. MCMULLEN.

[1] DISEASES OF THE SKIN. By Richard L. Sutton, M.D., Sc. D., LL.D. F.R.S. (Edin.). and Richard L. Sutton, Jr., A.M., M.D., L.R.C.P. (Edin.), 9th ed. St. Louis, Mo.: The C. V. Mosby Co., 1935.

[2] PHYSICAL DIAGNOSIS. By Warren P. Elmer, B.S., M.D., and W. D. Rose. 7th ed. St. Louis, Mo.: The C. V. Mosby Co., 1935.

THE manual[3] by Conybeare ranks with the best of the rather numerous manuals of diabetes that are available; its outstanding qualities are brevity and clearness. The author has outlined in 94 pages the general considerations which govern the physician's treatment of diabetes and, in an appendix of 22 pages, which may be obtained separately, he has provided for patients, detailed instructions with reference to practical points upon which successful self-care depends.

Detailed or technical discussions of the more theoretical and biochemical aspects of diabetic metabolism and a bibliography are intentionally omitted. The dangers of over-nutrition and excessive insulin dosage are stressed. By allowing 100 to 140 grams of carbohydrate the author believes that the diet can be made reasonably palatable and that special diabetic foods are not required. With such carbohydrate allowance he believes that there is no conclusive evidence that the amount of fat needed to furnish a maintenance diet predisposes to arteriosclerosis. The value of examinations of the urine four times daily (in the early morning, before luncheon, before dinner, and at bedtime) is emphasized.

To the physician who desires concise information as to modern conservative management of diabetes and to the intelligent patient this book will be helpful.

WALTER H. NADLER.

IN a ponderous volume[4] of 1028 pages of which 36 are index, Gradwohl apparently has attempted to produce an encyclopedia of clinical laboratory methods. Matters of technique are in fine print; interpretation and discussion of results are in larger type. "The standard technic of accepted procedures is given in strictest detail, together with an interpretation of all data obtained by standard tests," says the author in his preface. There is no attempt at selection and no definite recommendation of any one method. For example, 10 qualitative tests for albumin and 7 for sugar in the urine are described. The descriptions of technical procedures are models of brevity and are, usually, adequate and clear. In each case a constant formula is followed: The reagent and its preparation; the technique of the test; the changes that constitute a positive result with attention to possible misleading reactions. In those tests in which a quantitative result is the goal, the formula and a concrete example of its application are given.

A chapter on blood chemistry of 80 pages contains a useful table of the composition of normal human blood with the diseases or pathological states in which changes in each constituent may be encountered. The author is especially interested in hematology and the chapter on this subject, which occupies 180 pages, is useful and stimulating. Special

[3] MANUAL OF DIABETES. By J. J. Conybeare, M.C., M.D. (Oxon.), F.R.C.P., London: Oxford University Press, 1935.

[4] CLINICAL LABORATORY METHODS AND DIAGNOSIS; A TEXTBOOK ON LABORATORY PROCEDURES WITH THEIR INTERPRETATION. By R. B. H. Gradwohl, M.D. St. Louis, Mo.: The C. V. Mosby Co., 1935.

attention is given to the technique and significance of the Schilling count. This volume contains sections on the diagnosis of rabies, tests for pregnancy and hypersensitiveness, chemical analysis of milk, identification of pathogenic fungi, the making of autogenous vaccines, etc. The following chapter headings give an idea of the comprehensiveness of this book: Parasitology and Exotic Pathology, 95 pages; Bacteriological Applications to Clinical Diagnosis, 155 pages; Serology, 49 pages; Basal Metabolism, 17 pages; Postmortem Examinations, 41 pages; Tissue Cutting and Staining, 35 pages; Preparation of Museum Specimens, 16 pages; and Toxicologic Technique, 36 pages.

There are 324 illustrations in black and white, and 24 colored plates. Numerous tables and charts are useful adjuncts to the text. The declared purpose of the author in writing this volume was "to help the clinician, the laboratory worker, and the medical student to learn laboratory diagnosis." The book is larger and more comprehensive than is necessary for medical students, but it should be a very useful addition to the working library of any active laboratory.

J. P. SIMONDS.

THE general plan of the fifth edition of Clendening's book, *Methods of Treatment*,[1] has not been changed from that of his first edition. The book is still planned to furnish an outline of all methods of treatment in internal medicine. The revisions made represent the author's own ideas entirely in references to a given drug, diet, or form of treatment, etc.

The book is divided into two parts. Part I is devoted to general therapeutics, the methods used in treatment. In this section rest, drugs, diet, and hydrotherapy are discussed. The author describes in detail the method of preparation and the procedure to follow in carrying out the various forms of treatment.

Part II discusses special therapeutics, the application of therapeutics to particular diseases. The author gives full description to the application and the results to be expected from the various treatments he has outlined.

There are numerous illustrations throughout the book, showing instruments, describing technique, and a method of procedure for the treatments outlined.

This book, because it is a combination of materia medica and therapeutics, should be a valuable reference in any physician's library.

CLIFFORD J. BARBORKA.

THE excellent textbook[2] on urology by Eisendrath and Rolnick has been revised to include the more recent advances in diagnostic and therapeutic urology. An important addition is in the chapter on renal calculi, covering in a most complete manner the differential diagnosis of cholelithiasis. Transurethral electro-resection of various types of bladder neck obstructions has been considered in the light of the pathological anatomy and pathological physiology of prostatic hypertrophy. The chapters on anomalies of the kidneys and ureters, injuries of the kidney, and cystic disease of the kidneys are especially commendable. No other similar text that I have seen is so ably and adequately illustrated. While the work is somewhat too gross for the average medical student during his academic career, it is one that could well be used for reference during his school days and later found most useful during his years of clinical application.

VINCENT J. O'CONOR.

IN the second edition of Copeman's book,[3] which first appeared in 1933, many revisions and additions, particularly in the section on therapy, have been made. The use of gold salt therapy, histamine, bee venom, intravenous calcium, and short wave diathermy is discussed. The book is a general survey of the various rheumatic diseases. Chapters are devoted to the discussion of acute rheumatic fever, chorea, acute muscular rheumatism, and fibrositis, sciatica and neuritis, rheumatoid arthritis, osteoarthritis, and spondylitis. The treatment of these various afflictions is discussed in some detail in Part III which makes up two-thirds of the book. A chapter is devoted to prognosis. The author draws from his own rich experience, particularly in evaluating the various therapeutic measures. The book is very readable and should be of great value to the man in general practice.

J. ROSCOE MILLER.

IN a 202 page monograph[4] Guggisberg covers the subject of vitamins with special reference to obstetrics and gynecology as well as the field in general. The work is an enlargement and revision of his contribution as it originally appeared in Halban-Seitz, *Biologie und Pathologie des Weibes*, in 1929. The subject material is evenly divided. There is a general chapter on vitamins and more specifically the relation of vitamins to pregnancy, labor, the puerperium, the newborn, their relation to the sex organs, and internal secretions. Various sub-chapters of general biologic interest are included, such as relation to growth, constitution, nutrition, malignant tumors, tuberculosis, and wound healing. There is much repetition in the reading matter and much that is given is not new.

The author dislikes the alphabetical nomenclature of vitamins and suggests that they be named according to physical, chemical, and physiological properties. He follows out the classification of Winterstein and Funk, based largely on fat and water solubility. The vitamins of special interest in obstetrics and gynecology are the B-complex consisting of B_1 and

[1] METHODS OF TREATMENT. By Logan Clendening, M.D. 5th ed. St. Louis, Mo.: The C. V. Mosby Co., 1935.

[2] TEXT-BOOK OF UROLOGY; FOR STUDENTS AND PRACTITIONERS. By Daniel N. Eisendrath, M.D., and Harry C. Rolnick, M.D. 3d ed. rev. Philadelphia: J. B. Lippincott & Co., 1935.

[3] THE TREATMENT OF RHEUMATISM IN GENERAL PRACTICE. By W. S. C. Copeman, M.A., M.B., B.Ch. (Cantab.), M.R.C.P. (London). With a foreword by Sir William Hale-White, K.B.E., M.D., F.R.C.P., Hon. LL.D. 2d ed. Baltimore: William Wood & Co., 1935.

[4] DIE BEDEUTUNG DER VITAMINE FUER DAS WEIB. By Prof. Dr. Hans Guggisberg. Berlin and Vienna: Urban and Schwarzenberg, 1935.

B_2, C, D, and E. There are many references to experimental work, but the author cautions the reader in drawing conclusions from such work and applying them directly to the human species. It appears that, if there is a congenital factor as regards vitamins, the deficiency exists merely from birth for such factors were not supplied during pregnancy. Further disturbances in growth are aggravated only when the deficiency is continued. Reactions, however, are reversible. It is interesting to note in this connection that there is no fetal rickets, but if there is a lack of vitamin D supplied by the mother, a rachitic tendency in the newborn child could very quickly develop. The author is familiar with all of the world's literature, in fact has contributed much to the subject from his own laboratory. An excellent bibliography, including the world's literature from 1929 to date, is appended. W. B. SERBIN.

THE book by Colwell entitled *The Method of Action of Radium and X-rays on Living Tissues*[1] meets a need and it may be said at the outset that it succeeds in bridging the gap which it intends to bridge. It is surprising how well the author covers a tremendous field of experimental work in this comparatively small volume. His own experience in this field, based on the contributions made by himself and in co-operation with Sydney Russ, enables him to review the material very critically.

All branches of biology, biological chemistry, histology, serology, and clinical treatment, which are connected with the action of radiation on living tissues, are considered. In the first chapter the author gives a review of the effect of radiation on the cell. He considers the changes produced by radiation on different cell organs (cell membrane, cytoplasm, cytoplasmic inclusions, and nuclei). In this chapter he reviews the observations and interprets them from the point of view of the most modern knowledge of colloidal chemistry of the cell.

The second chapter covers the observations made on the chemical action of radiation. Here he considers the effect of rays on colloids, on oxidation and reduction phenomena as far as they are related to biological reactions. He also reports in this chapter the effect of radiation on enzymes. A large part is devoted to the effect of radiation on autolysis. The effect of rays on lipoids of the cell and on cholesterol is also reported. Special consideration is given to glutathione because of its fundamental importance in cell oxidation. This is followed by the report of Crabtree's work on the effect of radium radiation on tissue oxidation and carbohydrate metabolism. A short discussion of the results of radiation on the chemical composition of proteins is included.

In further chapters the action of radiation on the reticulo-endothelial system and some immunity reactions are reported. The effect of radiation on antibody formation and its dependence on the quality and dosage of the radiation are reported. These considerations constitute a basis for the important chapters which deal with the *effect of radiation on tumor growth;* since the immunity reactions are at least partly due, on the one hand, to the reticulo-endothelial system and, on the other hand, some careful investigations support the assumption that tumor growth is regulated by the function of the reticulo-endothelial system.

In the fifth chapter, "Radiation and Resistance to Tumor Growth," these questions are discussed extensively. The effect of radiation on tumor growth is considered from the point of view of the effect of radiation on the isolated cells; this means on tissue cultures *in vitro* before grafting these cells. Furthermore, the effect on tumor cells in animals and the effect on the reticulo-endothelial system of the bearer of the tumor are reported. It is particularly important from the clinical point of view that there are also experimental observations which indicate that the different amounts of radiation in the cells produce different results; so that a certain amount produces immunity reactions against further tumor growth (which may be due to certain contents of the tumor cells), whereas too large amounts of radiation seem to destroy these substances. The question of the importance of the reticulo-endothelial system for the growth of tumor metastases or for the resistance against this growth is critically discussed.

While this chapter deals mostly with grafted tumors, the last chapter, "The Action of Radiation on Malignant Tumors," gives the results of radiation effect on malignant tumors in man. It is based primarily on the classical work of Lacassagne and Monod, which is extensively reported, and the possibility of explaining the results on the basis of present knowledge is discussed. In logical connection with this material, the question of radiosensitivity of tumor tissues is considered.

In reporting this extensive material and considering the many special questions, the author never loses the broad general biological view. He attempts to show what clinical application can be made of this material. In this manner the book gives an excellent review of the present status of all questions which are related to radiotherapy, giving a safe, non-speculative basis for the application of experimental knowledge to clinical treatment.

This work serves as a basis for new experimental work, but it is more than a convenient guide for the experimental worker. It gives to the clinical radiologist the means of understanding and interpreting some of his observations and gives him new stimulation. For this reason, the book should be read by every radiologist who wishes to administer his treatment on a sound and intelligent basis.

MAX CUTLER.

[1] THE METHOD OF ACTION OF RADIUM AND X-RAYS ON LIVING TISSUES. Hector A. Colwell, M.B., Ph.D., M.R.C.P., D.P.H. London: Oxford University Press, 1935.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

THE SUBNORMAL MIND. By Cyril Burt, M.A., D.Sc. (Oxon.). London: Oxford University Press, 1935.

ESTUDOS CIRURGICOS. By Eurico Branco Ribeiro. São Paulo, Brasil: Sociedade Editora Medica Limitada, 1934.

THE DISEASES OF THE ENDOCRINE GLANDS. By Hermann Zondek, M.D. (Berlin). 3d. ed. rev. and enl., translated by Carl Prausnitz, M.D. (Breslau), M.R.C.S. (Eng.), L.R.C.P. (Lond.). Baltimore: William Wood & Co., 1935.

THE INTERNATIONAL MEDICAL ANNUAL; A YEAR BOOK OF TREATMENT AND PRACTITIONER'S INDEX. 1935, Fifty-third year. Baltimore: William Wood & Co., 1935.

CLINICAL TUBERCULOSIS. Edited by Benjamin Goldberg, M.D., F.A.C.P., F.A.P.H.A. Vols. 1 and 2. Philadelphia: F. A. Davis Co., 1935.

NATIONAL MEDICAL MONOGRAPHS. OBSTETRICS FOR THE GENERAL PRACTITIONER. By J. P. Greenhill, B.S., M.D., F.A.C.S. Edited by Morris Fishbein, M.D. New York: National Medical Book Co., Inc., 1935.

GYNECOLOGICAL AND OBSTETRICAL TUBERCULOSIS. By Edwin M. Jameson, B.S., M.D. Philadelphia: Lea & Febiger, 1935.

A SYNOPSIS OF REGIONAL ANATOMY. By T. B. Johnston, M.B., Ch.B. 3d ed. Philadelphia: Lea & Febiger, 1935.

THE PRINCIPLES AND PRACTICE OF SURGICAL NURSING. By Charles D. Lockwood, A.B., M.D., F.A.C.S., and John A. Wolfer, M.D., F.A.C.S. In collaboration with Mildred E. Newton, B.S., R.N. 2d rev. ed. New York: The Macmillan Co., 1935.

THE THEORY AND PRACTICE OF ANÆSTHESIA. By M. D. Nosworthy, M.A., M.D., B.Ch. (Cantab.) with a foreword by I. W. Magill. London: Hutchinson Scientific, 1935.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. A COMPLETE DICTIONARY OF THE TERMS USED IN MEDICINE, SURGERY, DENTISTRY, PHARMACY, CHEMISTRY, NURSING, VETERINARY SCIENCE, BIOLOGY, MEDICAL BIOGRAPHY, ETC., WITH THE PRONUNCIATION, DERIVATION, AND DEFINITION. By W. A. Newman Dorland, A.M., M.D., F.A.C.S. 17th ed. rev. and enl. With the collaboration of E. C. L. Miller, M.D. Philadelphia and London: W. B. Saunders Co., 1935.

NEW AND NONOFFICIAL REMEDIES, 1935; CONTAINING DESCRIPTIONS OF THE ARTICLES WHICH STAND ACCEPTED BY THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION ON JANUARY 1, 1935. Chicago: American Medical Ass., 1935.

ANNUAL REPRINT OF THE REPORTS OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR 1934 WITH THE COMMENTS THAT HAVE APPEARED IN THE JOURNAL. Chicago: American Medical Ass., 1934.

HUGH OWEN THOMAS; HIS PRINCIPLES AND PRACTICE. By D. McCrae Aitken, M.A., M.B., Ch.B. (Ed.), F.R.C.S. (Ed. and Eng.). London: Oxford University Press, 1935.

THE MEDICINE-MAN OF THE AMERICAN INDIAN AND HIS CULTURAL BACKGROUND. By William Thomas Corlett, M.D., L.R.C.P. (Lond.) Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1935.

DISEASES OF THE THYROID GLAND. By Arthur E. Hertzler, M.D. With a chapter on Hospital Management of Goiter Patients by Victor E. Chesky, M.D. 3d rev. ed. St. Louis, Mo.: The C. V. Mosby Co., 1935.

THE MEDICAL ANNUAL GENERAL INDEX AND REVIEW; FOR THE TEN YEARS 1925 TO 1934. Baltimore: William Wood & Co., 1935.

HORMONE DES OVARIUMS UND DES HYPOPHYSENVORDERLAPPENS; UNTERSUCHUNGEN ZUR BIOLOGIE UND KLINIK DER WEIBLICHEN GENITALFUNKTION. By Dr. Bernhard Zondek. 2d enl. ed. Wien: Julius Springer, 1935.

THE LIVERPOOL MEDICO-CHIRURGICAL JOURNAL. HUGH OWEN THOMAS CENTENARY NUMBER. Vol. 43, part 1, 1935. Liverpool: Medical Institution; London: H. K. Lewis & Co., Ltd.

THE CAROTID SINUS AND THE CEREBRAL CIRCULATION; AN ANATOMICAL, EXPERIMENTAL, AND CLINICAL INVESTIGATION. By Erik Ask-Upmark. Supplement 6 to ACTA PSYCHIATRICA ET NEUROLOGICA. Copenhagen: Levin & Munksgaard, 1935.

NUTRITION OF MOTHER AND CHILD. By C. Ulysses Moore, M.D., M.Sc. (Ped.), F.A.C.P. 4th rev. ed. Philadelphia and London: J. B. Lippincott Co., 1935.

DISEASES OF THE LIVER, GALL BLADDER, DUCTS AND PANCREAS; THEIR DIAGNOSIS AND TREATMENT. By Samuel Weiss, M.D., F.A.C.P. New York: Paul B. Hoeber, Inc., 1935.

A TEXT-BOOK OF FRACTURES AND DISLOCATIONS; COVERING THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT. By Kellogg Speed, S.B., M.D., F.A.C.S. 3d ed., thoroughly rev. Philadelphia: Lea & Febiger, 1935.

AGENTS OF DISEASE AND HOST RESISTANCE, INCLUDING THE PRINCIPLES OF IMMUNOLOGY, BACTERIOLOGY, MYCOLOGY, PROTOZOOLOGY, PARASITOLOGY AND VIRUS DISEASES. By Frederick P. Gay. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1935.

TRANSACTIONS OF THE SOUTHERN SURGICAL ASSOCIATION. VOL. 47, FORTY-SEVENTH ANNUAL MEETING. Edited by Alton Ochsner, M.D. New York: Paul B. Hoeber, Inc., 1935.

HIDATIDOSI ÓSEA. By Dr. Oscar Ivanissevich. Buenos Aires: Imprenta Amorrortu, 1934.

LA BRONCOGRAFÍA; EN EL ESTUDIO DE LAS AFECCIONES DEL TÓRAX. By Raul Piaggio Blanco and Federico Garcia Capurro. Montevideo: Imp. "El Siglo Ilustrado," 1935.

THE PNEUMONOKONIOSES (Silicosis). LITERATURE AND LAWS OF 1934. INTERNATIONAL ABSTRACTS, EXTRACTS AND REVIEWS OF THE PNEUMONOKONIOSES AND THEIR ASSOCIATED DISEASES AND SUBJECTS. By George G. Davis, M.D., Ella M. Salmonsen, and Joseph L. Earlywine. Chicago: Chicago Medical Press (Not Inc.), 1935.

PREVENTIVE MEDICINE AND HYGIENE. By Milton J. Rosenau. 6th ed. New York and London: D. Appleton-Century Co., 1935.

HUMAN PATHOLOGY; A TEXTBOOK. By Howard T. Karsner, M.D. With an introduction by Simon Flexner, M.D. 4th rev. ed. Philadelphia and London: J. B. Lippincott Co., 1935.

APHASIA; A CLINICAL AND PSYCHOLOGICAL STUDY. By Theodore Weisenburg, M.D., and Katharine E. McBride, Ph.D. New York: The Commonwealth Fund; London: Humphrey Milford—Oxford University Press, 1935.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

ROBERT B. GREENOUGH, Boston, *President* DONALD C. BALFOUR, Rochester, *President-Elect*

HOWARD C. NAFFZIGER, *Chairman;* THOMAS F. MULLEN, *Secretary, Committee on Arrangements*

PROGRAM FOR THE 1935 CLINICAL CONGRESS IN SAN FRANCISCO AND OAKLAND

CLINICAL CONGRESS PROGRAM IN BRIEF

Monday, October 28

9:00. Clinics in San Francisco hospitals.
9:30. Hospital conference—Fairmont Hotel, Ballroom.
2:00. Clinics in San Francisco hospitals.
2:00. Hospital conference—Fairmont Hotel, Ballroom.
2:00. Surgical films—Fairmont Hotel, Red Room.
5:00. State and Provincial Executive Committees—Fairmont Hotel, Gray Room.
8:15. Presidential meeting—War Memorial Opera House.

Tuesday, October 29

9:00. Clinics in San Francisco hospitals.
9:30. Section on ophthalmology and otolaryngology, scientific session—Fairmont Hotel, Gray Room.
9:30. Hospital conference—Fairmont Hotel, Ballroom.
10:00. Surgical films—Fairmont Hotel, Red Room.
2:00. Clinics in San Francisco hospitals.
2:00. Hospital conference—St. Mary's Hospital.
2:00. Surgical films—Fairmont Hotel, Red Room.
2:00. Fracture conference—Fairmont Hotel, Ballroom.
8:00. Hospital conference—St. Mary's Hospital.
8:15. Scientific session, general surgery—Auditorium, Veterans' Building.

Wednesday, October 30—Oakland Day

9:00. Clinics in Alameda County hospitals.
9:30. Hospital conference—Fairmont Hotel, Red Room.
9:30. Surgical films—Fairmont Hotel, Ballroom.
2:00. Clinics in Alameda County hospitals.
2:00. Surgical films—Fairmont Hotel, Ballroom.
2:00. Hospital conferences—University of California Hospital and San Francisco Hospital.
8:00. Community health meeting—Exposition Auditorium.
8:15. Scientific session, general surgery—Auditorium, Veterans' Building.

Thursday, October 31

9:00. Clinics in San Francisco hospitals.
9:30. Section on ophthalmology and otolaryngology, scientific session—Fairmont Hotel, Gray Room.
9:30. Hospital conference—Providence Hospital, Oakland.
10:00. Surgical films—Fairmont Hotel, Red Room.
1:30. Annual meeting—Fairmont Hotel, Ballroom.
2:00. Clinics in San Francisco hospitals.
2:00. Hospital conference—Samuel Merritt Hospital, Oakland.
3:00. Cancer symposium—Fairmont Hotel, Ballroom.
8:15. Scientific session, general surgery—Auditorium, Veterans' Building.

Friday, November 1

9:00. Clinics in San Francisco hospitals.
9:30. Section on ophthalmology and otolaryngology, scientific session—Fairmont Hotel, Gray Room.
10:00. Surgical films—Fairmont Hotel, Red Room.
10:00. Hospital conference and tour of Alameda County hospitals.
11:00. Meeting of new Fellows, class of 1935—Fairmont Hotel, Ballroom.
2:00. Clinics in San Francisco hospitals.
2:00. Surgical films—Fairmont Hotel, Red Room.
2:00. Conference on industrial medicine and traumatic surgery—Fairmont Hotel, Ballroom.
8:15. Convocation—Scottish Rite Auditorium.

THE surgeons of San Francisco and Oakland have prepared a program of clinics and demonstrations that will provide a complete showing of the clinical activities in all departments of surgery in that great medical center on the Pacific coast during the twenty-fifth annual Clinical Congress of the American College of Surgeons, October 28–November 1. The Committee on Arrangements has been assured of the hearty co-operation of the clinicians at the two medical schools and twenty-seven hospitals that will participate in the program.

The clinical program, published in the following pages, will be further revised and amplified during the weeks preceding the Congress. Operative clinics and demonstrations in the hospitals are scheduled for the morning and afternoon of Monday, October 28, beginning at 9 o'clock and for the mornings and afternoons of each of the four following days. The real program of the Congress will be published daily—a complete and accurately detailed program that will be posted in the form of bulletins at headquarters each afternoon for the succeeding day. The same material will be issued in printed form the following morning.

Special features of the clinical program include: (1) Cancer clinics demonstrating the treatment of cancer by surgery, radium and X-ray; (2) fracture clinics demonstrating modern methods of treatment; (3) clinics in traumatic surgery demonstrating the newer methods of rehabilitation of injured patients by surgery and physiotherapy.

During the Congress the hospital ship *Relief* of the U. S. Navy will be docked at a convenient point on the waterfront. Clinics on board ship will be given on Tuesday morning by Commander Horace R. Boone and associates, and arrangements will be made for an inspection of the ship by the visiting surgeons and their friends during the Congress.

SPECIAL PROGRAMS AT HEADQUARTERS

A conference on fractures, under the auspices of the College Committee on the Treatment of Fractures, will be held in the Gold Ballroom of the Fairmont Hotel on Tuesday afternoon at 2 o'clock. A detailed program is presented in the following pages.

Under the auspices of the College Committee on the Treatment of Malignant Diseases a symposium on cancer will be presented in the Gold Ballroom of the Fairmont Hotel on Thursday afternoon following the annual meeting of Fellows. The symposium will concentrate on the standard accepted methods for the treatment of cancer. A report on five-year results of therapy, compiled by the Department of Clinical Research continuing the gathering of statistics on this subject from clinicians in various parts of the United States and Canada during the past several years, will be included. A detailed program is presented in the following pages.

The annual conference of the Board on Industrial Medicine and Traumatic Surgery will be held in the Gold Ballroom of the Fairmont Hotel on Friday afternoon at 2 o'clock. The program, which appears in the following pages, includes a report of the activities of the College in conducting investigations and surveys among industrial establishments during the past year in continuation of the work done along these lines during the past five years.

EVENING MEETINGS

Programs for the five evening sessions, as prepared by the Central Executive Committee, are presented in the following pages. At the presidential meeting on Monday evening in the War Memorial Opera House following the address by the retiring president, Dr. Robert B. Greenough, of Boston, the new officers elected at the 1934 Clinical Congress will be inaugurated—Dr. Donald C. Balfour, Rochester, Minn., president; Dr. Arthur W. Allen, Boston, and Dr. John A. Gunn, Winnipeg, vice-presidents. Dr. George Crile, of Cleveland, Chairman of the Board of Regents, will deliver the annual oration on surgery, his subject being "The American College of Surgeons —Past, Present and Future." This address will include a tribute to the life and work of the late Dr. Franklin H. Martin.

Scientific sessions will be held in the Auditorium of the Veterans' Building on Tuesday, Wednesday and Thursday evenings, at which eminent surgeons of the United States and Canada will present and discuss papers on surgical subjects of timely importance.

The annual convocation of the College will be held on Friday evening in the Scottish Rite Auditorium, at which the 1935 class of candidates will be received into Fellowship. The president, Dr. Donald C. Balfour, of Rochester, Minn., will deliver his inaugural address on this occasion, and Dr. Robert Gordon Sproul, B.S., LL.D., president of the University of California, will deliver the Fellowship address.

OPHTHALMOLOGY AND OTOLARYNGOLOGY

The committee in charge of the section on surgery of the eye, ear, nose and throat has arranged a program of ophthalmological and otolaryngological clinics and demonstrations at the hospitals and medical schools, as published in the following pages. Also, scientific sessions are to be held at the Fairmont Hotel on Tuesday, Thursday and Friday mornings, at which clinical papers are to be presented by visiting surgeons and discussed by local clinicians. The programs for these scientific sessions appear in the following pages.

MEETING OF NEW FELLOWS

Candidates for fellowship in the American College of Surgeons, class of 1935, will assemble in the Gold Ballroom of the Fairmont Hotel at 11 a.m. on Friday for the necessary instructions previous to receiving their fellowships and to sign the fellowship roll.

COMMUNITY HEALTH MEETING

Following its established custom and in recognition of an obligation to the public to provide authoritative information on modern surgery, better hospitals, and the prevention of disease, a community health meeting will be held on Wednesday evening at 8 o'clock, under the auspices of the American College of Surgeons, in the Exposition Auditorium. A program appropri-

ate for the occasion consisting of brief interesting talks on scientific medicine, health, and hospitals, including a motion picture film on a medical subject of interest to the laity, will be presented, as follows:

DONALD C. BALFOUR, M.D., Rochester, Minnesota, presiding.
Invocation.
Address of Welcome. HOWARD C. NAFFZIGER, M.D., San Francisco, Chairman, Committee on Arrangements.
Message of Greeting. JACOB C. GEIGER, M.D., San Francisco, Director of Public Health.
The American College of Surgeons—Its Aims and Objects. GEORGE CRILE, M.D., Cleveland.
The March of Scientific Medicine. ALLEN B. KANAVEL, M.D., Chicago.
The Approved Hospital—What It Means to You (Illustrated). MALCOLM T. MACEACHERN, M.D., Chicago.
Prevention of Heart Disease. LEROY LONG, M.D., Oklahoma City, Okla.
The Course and Curability of Cancer (Illustrated). BOWMAN C. CROWELL, M.D., Chicago.
Maternal Care. C. JEFF MILLER, M.D., New Orleans.
The Conservation of the Health Welfare of Workers in Industry (Illustrated). FREDERIC A. BESLEY, M.D., Waukegan, Ill.
Patients, Doctors, and Hospitals. ROBERT JOLLY, Houston, Texas.
Medical motion picture film.

OAKLAND DAY

Wednesday, October 30, will be designated as Oakland Day. The clinical program for that day will be transferred to the hospitals in Alameda County on the east side of the bay, which includes Oakland, Berkeley and Alameda—a great metropolitan area with a population of over 500,000 people, having many splendid hospitals and the great University of California in Berkeley. No clinics will be scheduled in San Francisco hospitals on that day. Besides a wealth of clinical facilities, there are many points of interest which it will be the pleasure of the surgeons living in the district to show to the visiting Fellows and their families. The Oakland committee has arranged special transportation facilities for the visiting Fellows and their friends.

HOSPITAL CONFERENCE

The eighteenth annual hospital standardization conference will open with a session in the Gold Ballroom of the Fairmont Hotel at 9:30 on Monday morning at which addresses will be delivered by distinguished representatives of several organizations. At this session the annual report of the hospital standardization activities of the College for 1935, including the list of approved hospitals, will be presented.

Monday afternoon will be given over to a discussion of hospital problems from various aspects in which administrators, surgeons and others will participate.

Tuesday morning will be devoted to a "panel" discussion of the hospital's obligation to its community and its part in the community life professionally and economically.

A joint session with the Association of Record Librarians will be held on Wednesday morning at which problems concerning hospital records are to be discussed.

Tuesday and Wednesday afternoons will be devoted to definitely planned departmental demonstrations in hospital administration in certain of the San Francisco hospitals. These demonstrations will show how various departments function, affording those present an excellent opportunity to see how other institutions carry on their activities.

Thursday will be Oakland Hospital Day and a program of demonstrations of special interest will be carried on in four of the major hospitals in Alameda County with the co-operation of the Oakland Hospital Council. Many new features and procedures in hospital management will be demonstrated.

Through the courtesy of Dr. D. W. Black, medical director of the Highland Hospital and director of Alameda County health activities, those who are interested in the Alameda plan for medical and hospital care will be given an opportunity to make an excursion through the county on Friday.

STATE AND PROVINCIAL COMMITTEES

A meeting of the State and Provincial Executive Committees with officers of the College has been called for 5 o'clock Monday afternoon in the Gray Room of the Fairmont Hotel. This meeting is called for the purpose of obtaining information on which may be based the itinerary of the College for its sectional meetings and the desirable grouping of the states and provinces.

ANNUAL MEETING

The annual meeting of the Fellows of the College will convene in the Gold Ballroom of the Fairmont Hotel at 1:30 o'clock Thursday afternoon. Reports by the officers and chairmen of the standing committees on the various activities of the College will be presented, followed by the election of officers.

SURGICAL FILM EXHIBITIONS

Throughout the week surgical motion picture films, both sound and silent, will be exhibited daily in the Red Room of the Fairmont Hotel. This showing of films, demonstrating clinical

features of interest, has met with popular acceptance in recent years. A number of new films are to be shown. Detailed programs will appear in the Daily Bulletin.

HEADQUARTERS—TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Fairmont and Mark Hopkins hotels. At the former the Terrace Ballroom and Foyer, the Gold Ballroom and other large rooms on the main floor and on the terrace have been reserved for scientific sessions and conferences, registration and clinic ticket bureaus, bulletin boards, exhibits, executive offices, etc.

The Technical Exhibition, including the registration and clinic ticket bureaus, will be located in the ballroom and foyer on the terrace floor of the Fairmont Hotel. In these rooms will also be found the bulletin boards on which the daily clinical program will be posted each afternoon. The leading manufacturers of surgical instruments, X-ray apparatus, operating room lights, hospital apparatus and supplies, ligatures, dressings, pharmaceuticals and publishers of medical books will be represented in this exhibition.

A VACATION ON THE PACIFIC COAST

Many Fellows of the College and their guests, who will attend the Clinical Congress in San Francisco, are planning to take advantage of the opportunity which naturally suggests itself to include a vacation and sight-seeing trip on the Pacific coast. All the railways are offering very low round-trip rates with liberal stopover privileges from all parts of the United States and Canada, with the added privilege of traveling to the coast by one route and returning by another, thereby affording unusual opportunities for visiting many points of interest en route.

Few cities have so impressive a setting as San Francisco. The rugged hills upon which it is built, the Golden Gate, Mt. Tamalpais, the misty bay to the east, Oakland and Berkeley, and the hills beyond, all contribute to its grandeur. It is a pleasing picture and the citizens of San Francisco have not been unmindful of it. Their parks and residence districts have a dignity and beauty in keeping with the scene about them.

Along the Pacific coast one will find much to claim his attention. First, there is the California-Pacific International Exposition at San Diego—a World's Fair of magnitude, with many important medical and other scientific exhibits—erected in Balboa Park, a tropical fairyland. The buildings, which are permanent structures, have their dignity and beauty enhanced by the gorgeous setting. Sunday, November 3, has been designated as American College of Surgeons Day, and a special program for that day is being prepared.

Los Angeles with its mountain background, its broad boulevards, its nearby beaches, its hospitality—and Hollywood—afford opportunities for entertainment and recreation.

Traveling south but a short distance from San Francisco, one enters the San Joaquin valley at Merced—the gateway to Yosemite National Park, where one views one of the most famous spectacles of nature. The valley occupies but eight square miles, but in this limited space are gathered more natural wonders than can be found in any other similar area in the world. It contains eight waterfalls, the lowest twice the height of Niagara, and the highest the equal of ten Niagaras. There are meadows of deep grass and wild flowers, a rushing mountain river, forests and towering cliffs.

Del Monte and Pebble Beach, on the famous seventeen mile drive, are but a short distance from San Francisco. Other nearby points of interest are Paso Robles, with its almond groves, lovely Santa Barbara with its famous Old Mission, and a score of cities whose names are associated with the romantic Spanish period—a fairyland of natural and cultivated beauty.

To the north there are many cities to claim your attention: Vancouver, in British Columbia, built up against mighty mountains with a primeval forest almost at its environs; Seattle, the great shipping center of the northwest, with Puget Sound to the west and beyond it the white-capped Olympics; in the distance the Cascade Mountains and looming to the southeast, Mt. Rainier, held in special veneration by the Indians as "the mountain that was God"—no peak in the United States is so majestic; Portland, famous for its natural beauty—a literal bower of roses; the blue miracle of Crater Lake, Mt. Shasta and Lassen Peak, the only active volcano in the United States.

No matter what route one takes he will view a truly great and inspiring American scene—the thriving farm lands of the midwest, the noble stretches of prairie, the sublime mountain ranges of the Rockies, the grandeur of the desert, the towering forests of the far west, and then the blue Pacific.

This year one will travel in comfort and luxury undreamed of a short time ago. Whatever route one travels he will find his train air-conditioned. This single factor adds so much to the comfort and pleasure of a transcontinental journey that it definitely and certainly marks the beginning of a new era in transportation history.

RAILWAY FARES FROM VARIOUS CITIES TO SAN FRANCISCO AND RETURN

Low round-trip fares will be in effect from all points in the United States and Canada. Consult your local ticket agent as to exact fare. Summer tourist tickets are good going via one route and returning via the same or any other authorized route, with a final return limit of November 30.

	Round Trip Fare	Lower Berth One Way
Atlanta	$100.75	$21.25
Baltimore	120.75	24.00
Boston	132.80	25.88
Buffalo	109.55	21.38
Chicago	86.00	15.75
Cincinnati	97.00	19.50
Cleveland	101.35	19.50
Detroit	98.30	19.50
Duluth	86.00	17.75
Kansas City	72.00	13.25
Memphis	85.15	14.50
Minneapolis	86.00	15.75
Montreal	122.55	24.75
New York	126.90	24.75
New Orleans	85.15	14.00
Omaha	72.00	13.25
Philadelphia	122.85	24.00
Pittsburgh	107.10	20.25
Portland, Ore	29.70	5.50
St. Louis	81.50	15.00
St. Paul	86.00	15.75
Seattle	37.05	6.75
Toronto	108.15	21.38
Vancouver, B. C	42.55	8.75
Washington	120.75	24.00
Winnipeg (via Portland)	86.00	18.75

ADVANCE REGISTRATION

The hospitals and medical schools of San Francisco and Oakland afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to a number that can be comfortably accommodated at the clinics—the limit of attendance being based upon the result of a survey of the amphitheatres, operating rooms, and laboratories of the hospitals and medical schools to determine their capacity for visitors. It is expected, therefore, that those surgeons who wish to attend the Congress will register in advance.

Admittance to all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the distribution of the visiting surgeons among the several clinics and insures against overcrowding, as the number of tickets issued for any clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.

SAN FRANCISCO HOTELS AND THEIR RATES

In addition to the two headquarters hotels—the Fairmont and Mark Hopkins—there are a number of first-class hotels within short walking distance of headquarters providing ample hotel facilities at reasonable rates. The following hotels are recommended by the Committee:

	Minimum Rate with Bath	
	Single	Double
Bellevue, Geary and Taylor	$3.00	$4.00
Californian, Taylor and O'Farrell	3.00	4.50
Clift, Geary and Taylor	3.50	5.00
El Cortez, Geary near Taylor	3.00	4.50
Fairmont, Mason and California	3.50	5.00
Gaylord, Jones near Geary	3.00	4.00
Mark Hopkins, Mason and California	3.50	5.00
Palace, Market and New Montgomery	3.50	5.00
Plaza, Post and Stockton	3.00	4.00
Sir Francis Drake, Powell and Sutter	3.50	5.00
Stewart, 353 Geary	2.50	4.00
St. Francis, Union Square	3.50	5.00

OPHTHALMOLOGY AND OTOLARYNGOLOGY—SCIENTIFIC SESSIONS

Tuesday, 9:30—Gray Room, Fairmont Hotel

JOSEPH L. MCCOOL, M.D., Chairman.

A New Iridosclerectomy Operation for Glaucoma. CONRAD BERENS, M.D., New York.
Discussion opened by A. R. IRVINE, M.D., Los Angeles.

The Management of Complications in the Operation for Senile Cataract. JOHN O. MCREYNOLDS, M.D., Dallas, Texas.
Discussion opened by JOSEPH L. MCCOOL, M.D., San Francisco.

Orbital Abscess. DWIGHT C. ORCUTT, M.D., Chicago.
Discussion opened by RAYMOND J. NUTTING, M.D., Oakland.

The Surgical Approach to the Nasal Accessory Sinuses. WILLIAM MITTHOEFER, M.D., Cincinnati, Ohio.
Discussion opened by EDWARD DEWALL, M.D., San Francisco.

Motion Picture and Microscopical Studies of the Trans-antrum-ethmo-sphenoidal Operation. CHARLES E. FUTCH, M.D., Los Angeles.
Discussion opened by GEORGE MCCLURE, M.D., Oakland.

Thursday, 9:30—Gray Room, Fairmont Hotel

HARRINGTON B. GRAHAM, M.D., Chairman.

Brief Consideration of the History of the Development of Mastoidectomy. ROBERT SONNENSCHEIN, M.D., Chicago.
Discussion opened by COL. A. E. SCHLANZER.

Unique Symptoms and Effects of Sphenoidal Empyema. CHESTER H. BOWERS, M.D., Los Angeles.
Discussion opened by ROBERT C. MARTIN, M.D., San Francisco.

Surgical Correction of Defects Due to Paralysis of the Muscles of the Eyes and of the Eyelids. MEYER WIENER, M.D., St. Louis.
Discussion opened by RODERICK O'CONNOR, M.D., Oakland.

The Relation of Orthoptics to the Surgical Treatment of Comitant Strabismus. AVERY DEH. PRANGEN, M.D., Rochester, Minnesota.
Discussion opened by HANS BARKAN, M.D., San Francisco.

Friday, 9:30—Gray Room, Fairmont Hotel

JOSEPH L. MCCOOL, M.D., Chairman.

Separated Retina Treatment with Galvano Cautery. CLIFFORD B. WALKER, M.D., Los Angeles.
Discussion opened by DOHRMANN K. PISCHELL, M.D., San Francisco.

An Analysis of Five Hundred Intra-ocular Steel Operations. SIDNEY WALKER, JR., M.D., Chicago.
Discussion opened by OTTO BARKAN, M.D., San Francisco.

Motion Picture Study of Laryngeal Pathology. F. E. LEJEUNE, M.D., New Orleans.
Discussion opened by WALLACE B. SMITH, M.D., San Francisco.

Cancer of the Larynx. W. E. SAUER, M.D., St. Louis.
Discussion opened by HARRINGTON B. GRAHAM, M.D., San Francisco.

The Selection of Treatment for Malignancy of the Upper Respiratory Tract. FREDERICK A. FIGI, M.D., Rochester, Minnesota.
Discussion opened by REA E. ASHLEY, M.D., San Francisco.

PROGRAMS FOR EVENING MEETINGS

Presidential Meeting—Monday, 8:15 p.m.—War Memorial Opera House

Invocation.

Address of Welcome. HOWARD C. NAFFZIGER, M.D., San Francisco, Chairman of Committee on Arrangements.

Introduction of Foreign Guests.

Address of the Retiring President: The Conscience of the Surgeon. ROBERT B. GREENOUGH, M.D., Boston.

Inauguration of Officers: President, DONALD C. BALFOUR, M.D., Rochester, Minn.; First Vice-President, ARTHUR W. ALLEN, M.D., Boston; Second Vice-President, JOHN A. GUNN, M.D., Winnipeg.

Treatment of Peptic Ulcer, Based on Physiological Principles. ALTON OCHSNER, M.D., New Orleans.

Annual Oration on Surgery: The American College of Surgeons—Past, Present and Future. GEORGE CRILE, M.D., Cleveland.

Tuesday, 8:15 p.m.—Auditorium, Veterans Building

Symposium on Hypertension:

The Medical Problem and Management of Essential Hypertension. S. MARX WHITE, M.D., Minneapolis.

Surgery in its Relation to Hypertension. ALFRED W. ADSON, M.D., Rochester, Minn.

Treatment of Hypertension by Splanchnicectomy. MAX MINOR PEET, M.D., Ann Arbor, Mich.

Wednesday, 8:15 p.m.—Auditorium, Veterans Building

The Diagnosis and Treatment of Stone in the Common Duct. ARTHUR W. ALLEN, M.D., Boston.

Adrenal Cortical Tumors. GEORGE F. CAHILL, M.D., New York.

Some Aspects of Maternal Nutrition. LEIGHTON C. CONN, M.D., Edmonton, Alta.

Fracture Oration: Fundamentals versus Gadgets in the Treatment of Fractures. PAUL B. MAGNUSON, M.D., Chicago.

Thursday, 8:15 p.m.—Auditorium, Veterans Building

Ischemic Contractures. ARTHUR STEINDLER, M.D., Iowa City, Iowa.

Cerebral Injuries Due to External Trauma. GEORGE W. SWIFT, M.D., Seattle.

The Diagnosis of Endometrial Hyperplasia. LUCIUS E. BURCH, M.D., Nashville, Tenn.

The Iodine Relationships of Thyroid Disease. GEORGE M. CURTIS, M.D., Columbus, Ohio.

Convocation—Friday, 8:15 p.m.—Scottish Rite Auditorium

Invocation.

Presentation of Candidates for Fellowship.

Conferring of Fellowships. The President.

Conferring of Honorary Fellowships. The President.

President's Inaugural Address: The Trend of Surgery. DONALD C. BALFOUR, M.D., Rochester, Minn.

Fellowship Address: The Social Significance of Medicine. ROBERT GORDON SPROUL, B.S., LL.D., Berkeley, Calif.

CONFERENCES AND SYMPOSIA

FRACTURE CONFERENCE

Tuesday, 2 p.m.—Gold Ballroom, Fairmont Hotel

FREDERIC W. BANCROFT, M.D., New York, Chairman of Committee on Treatment of Fractures, presiding.
Highway First Aid Stations. E. PAYNE PALMER, M.D., Phoenix, Arizona.
Fender Fractures into the Knee Joint. FREDERIC J. COTTON, M.D., Boston.
Status of Fractures in the Field of Surgery. CLAY RAY MURRAY, M.D., New York
Local Anesthesia in Fractures. E. DUNBAR NEWELL, M.D., Chattanooga, Tenn.
The Causes of Non-Union; Bone Growth and Regeneration. WILLIAM R. CUBBINS, M.D., Chicago.
Open Treatment of Fractures of the Femur. GROVER C. WEIL, M.D., Pittsburgh.

CANCER SYMPOSIUM

Thursday, 3 p.m.—Gold Ballroom, Fairmont Hotel

CHARLES A. DUKES, M.D., Oakland, Calif., Chairman of Committee on the Treatment of Malignant Diseases, presiding.
Symposium: Standard Methods of Treatment of Cancer:
Prostate. BENJAMIN S. BARRINGER, M.D., New York.
Fundus Uteri. ALSON R. KILGORE, M.D., San Francisco.
Colon. THOMAS E. JONES, M.D., Cleveland.
Larynx. GORDON B. NEW, M.D., and F. A. FIGI, M.D., Rochester, Minn.
Melanoma. FRANK E. ADAIR, M.D., New York.
A Diagnostic Cancer Clinic in a Private Hospital. C. HIRAM WEAVER, M.D., Los Angeles.
Five-Year Results of Treatment. BOWMAN C. CROWELL, M.D., Chicago.

CONFERENCE ON INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY

Friday, 2 p.m.—Gold Ballroom, Fairmont Hotel

FREDERIC A. BESLEY, M.D., Waukegan, Ill., Chairman of Board on Industrial Medicine and Traumatic Surgery, presiding.
Silicosis and Its Control. R. R. SAYERS, M.D., and R. R. JONES, M.D., Washington.
Analysis of Five Hundred Intra-ocular Steel Operations. SYDNEY WALKER, JR., M.D., Chicago.
Tannic Acid and Silver Nitrate in Burns. ADALBERT G. BETTMAN, M.D., Portland, Ore.
Heat Exhaustion and Its Prevention. LOYAL A. SHOUDY, M.D., Bethlehem, Pa.
Immediate and Delayed Repair of Tendon Injuries. MICHAEL L. MASON, M.D., Chicago.
Report of the 1935 Survey of Medical Services in Industry by the American College of Surgeons. M. N. NEWQUIST, M.D., Chicago.

ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

Monday, 9:30–12:30—Gold Ballroom, Fairmont Hotel

ROBERT B. GREENOUGH, M.D., Boston, presiding.
Greetings. JACOB C. GEIGER, M.D., and LEON M. WILBOR, M.D., San Francisco.
The Hospital Standardization Movement of the American College of Surgeons and Announcement of Approved List of Hospitals for 1935. GEORGE CRILE, M.D., Cleveland.
The Hospital and the Changing Social Order. SISTER JOHN GABRIEL, R.N., Seattle, Wash.
Opportunities for the Training of Surgeons in the Approved Hospital. IRVIN ABEL, M.D., Louisville.
Organization and Administration of an Oxygen Therapy Service in a General Hospital. R. C. BUERKI, M.D., and RALPH WATERS, M.D., Madison, Wis.
An Accredited Pathologist for Every Approved Hospital. PHILIP HILLKOWITZ, M.D., Denver.
The Future of the Voluntary Hospital. ROBERT JOLLY, Houston, Texas.
Discussion. FREDERIC A. BESLEY, M.D., Waukegan, Ill.

Monday, 2:00–5:00—Gold Ballroom, Fairmont Hotel

BENJAMIN W. BLACK, M.D., Oakland, presiding.

Application of the Principles of Hospital Standardization from the Viewpoints of:

Hospital Trustee. W. C. CRANDALL, La Jolla, Calif.
Hospital Administrator. PAUL H. FESLER, Chicago.
Medical Staff Member. JACOB F. HIGHSMITH, M.D., Fayetteville, N. C.
Clinical Pathologist. ALVIN G. FORD, M.D., Pasadena, Calif.
Radiologist. EDWARD S. BLAINE, M.D., Los Angeles.
Nurse. SISTER MARY STEPHANIE, R.N., San Francisco.
Dietitian. LUCILE WAITE, San Leandro, Calif.
Medical Social Worker. MARGUERITE L. SPIERS, Oakland.
Medical Educator. ALEXANDER R. MUNROE, M.D., Edmonton, Alta.
Economist. DANIEL CROSBY, M.D., Oakland.

Tuesday, 9:30–12:30—Gold Ballroom, Fairmont Hotel

ARTHUR M. CALVIN, St. Paul, presiding.

Innovations in Hospital Equipment and Supplies from the Standpoint of Efficiency, Economy, and Service. G. W. OLSON, Los Angeles.

The Institutional Care of Chronic and Convalescent Patients. A. C. JENSEN, San Leandro, Calif.

Panel Discussion Conducted by JOSEPH G. NORBY, Minneapolis.

To Create Understanding—Public Relations. FRANK J. WALTER, Denver.
To Provide Adequate Service. G. WAITE CURTIS, San Francisco.
To Bring the Cost of Hospital Service Within Reach of the People Served. R. E. HEERMAN, Los Angeles.
To Provide Adequate Service to the Indigent Patient. J. V. BUCK, Spokane, Wash.
To Educate Nurses, Doctors, and Others Engaged in the Care of the Sick. CAROLYN E. DAVIS, Portland, Ore.

Tuesday, 2:00–5:00—St. Mary's Hospital

Demonstrations and round table discussions dealing with methods of standardization and administration. Conducted by SISTER MARY THOMASINE, Superintendent, and heads of departments.

Business administration—equipment, personnel, accounting, reports, hospital costs, methods for meeting present economic conditions.

Organization and management of food service—control and responsibility, personnel, types of service, special diets.

Pharmacy service—organization and management, physical requirements, personnel, accounting.

Adjunct departments—Organization and management of clinical laboratory, X-ray and physical therapy departments, technical service, routine examination, records and filing.

Tuesday, 8:00–10:00 p.m.—St. Mary's Hospital

Round Table Conference—A discussion of every-day hospital problems as applied to the professional and economic aspects of hospital administration. Conducted by ROBERT JOLLY, Houston, Texas.

Wednesday, 9:30–12:30—Gold Ballroom, Fairmont Hotel

Joint Session with Association of Record Librarians of North America. R. C. BUERKI, M.D., Madison, Wis., presiding.

Securing, Supervising, and Using Medical Records. MALCOLM T. MACEACHERN, M.D., Chicago.

Discussion from the viewpoints of:

Hospital Administrator. THEODORE E. SWARTZ, M.D., Oakland.
Physician. S. MARX WHITE, M.D., Minneapolis.
Surgeon. ALTON OCHSNER, M.D., New Orleans.
Obstetrician. D. G. TOLLEFSON, M.D., Los Angeles.
Ophthalmologist and Otolaryngologist. WILLIAM W. PEARSON, M.D., Des Moines, Iowa.
Record Librarian. RUTH M. SNIDER, Chicago.

Group Studies Essential to Scientific Efficiency. SISTER M. SERVATIA, R.N., Kansas City, Mo.

Wednesday, 2:00–5:00—University of California Hospital

Demonstration and round table conference on the organization and management of the medical records department. Conducted by F. S. DURIE, Superintendent, and heads of departments.

Physical requirements, personnel, supervision, filing and cross-indexing records, uses of records, special problems, standard nomenclature.

Wednesday, 2:00–5:00—San Francisco Hospital

Demonstrations and round table conference on care of the obstetrical patient in the general hospital. Conducted by LEON M. WILBOR, M.D., Superintendent, and heads of departments.

Prenatal care, admission procedure, care of the patient in labor, delivery room technique, postpartum care, the isolated or septic patient, care of the newborn, follow-up and end-results, review of morbidities and mortalities, records. Motion picture "Around the Clock with You and Your Baby."

OAKLAND HOSPITAL DAY—THURSDAY

10:00 a.m.—1:00 p.m.—Providence Hospital

Demonstrations and round table conferences:

Economies in hospital management. Staff of Providence Hospital.

Organization and management of the housekeeping department. Staff of Fairmont Hospital.

Organization and functioning of the social service department. Staffs of Alameda County and Berkeley General Hospitals.

2:00–5:00—Samuel Merritt Hospital

Demonstrations and round table conferences:

Admission and discharge procedures. Staffs of the Samuel Merritt and Peralta Hospitals.
Organization and management of a central supply room. Staff of Samuel Merritt Hospital.
Hospital formulary. Staff of Peralta Hospital.

Friday, 10 a.m.

Inspection of the emergency system of the Alameda County Hospital and tour of county institutions for study of the Alameda plan.

COMMITTEE ON ARRANGEMENTS

Executive Committee

Howard C. Naffziger, Chairman; Thomas F. Mullen, Secretary

Leroy C. Abbott	Charles A. Dukes	Frank Hinman	Frank W. Lynch
Harold Brunn	Leo Eloesser	Emile F. Holman	Joseph L. McCool
Edmund Butler	Oliver D. Hamlin	Alson R. Kilgore	Isaac W. Thorne
Whitfield Crane			

SUB-COMMITTEES—SAN FRANCISCO

Clinical Program Committee (General Surgery)—Alson R. Kilgore, Chairman; Edmund Butler, Thomas F. Mullen.

Clinical Program Committee (Eye, Ear, Nose and Throat Surgery)—Joseph L. McCool, Chairman; H. B. Graham, Warren D. Horner.

Transportation Committee—Emile F. Holman, Chairman; G. D. Delprat, Millard R. Ottinger.

Public Meeting Committee—Jacob C. Geiger, Chairman; Harold M. Behneman, Edgar L. Gilcreest, Jacques P. Gray, Leon M. Wilbor.

Golf Committee—Leroy Brooks, Chairman; Harry Alderson, James Morgan, Frank Sheehy.

HOSPITAL REPRESENTATIVES

SAN FRANCISCO—GENERAL SURGERY

Franklin Hospital—Ernst Gehrels, Leroy Brooks.

French Hospital—Asa Collins, Edmund Morrissey.

Hospital for Children—Leroy Abbott, Alma Pennington.

Letterman General Hospital—Major Frank C. Cole.

Mary's Help Hospital—Isaac Thorne, Raymond Millzner, Everett Carlson, Dudley Smith.

Mount Zion Hospital—Harold Brunn, Franklin Harris, Albert L. Brown.

St. Francis Hospital—James O'Connor, Calvin A. Walker.

St. Joseph's Hospital—Alson R. Kilgore, J. Minton Meherin.

St. Luke's Hospital—G. D. Delprat, Otto Pflueger.

St. Mary's Hospital—Thomas E. Bailly, Philip Arnot, Daniel Sooy.

San Francisco Hospital—Stanford University Service: Leo Eloesser, C. Mathewson, L. Rogers. University of California Service: Harold Brunn, C. Latimer Callander, George K. Rhodes.

Shriners' Hospital for Crippled Children—Sylvan Haas.

Southern Pacific General Hospital—William Washburn, Frank R. Girard.

Stanford University Hospitals—Emile Holman, Philip K. Gilman, Frederick Reichert.

Stanford University School of Medicine—Loren Chandler, Emmet Rixford.

United States Marine Hospital—Mark J. White, Richey L. Waugh.

University of California Hospitals—Howard C. Naffziger, H. Glenn Bell.

University of California Medical School—Langley Porter, Wallace Terry.

Veterans Administration Facility—P. E. Johnson, Berthel H. Henning, John A. Kennedy.

SAN FRANCISCO—SURGERY OF THE EYE, EAR, NOSE AND THROAT

French Hospital—Edward C. Fabre-Rajotte, Victor d'Ercole.

Hospital for Children—George Hosford.

Letterman General Hospital—A. E. Schlanser, H. C. Maxwell.

Mary's Help Hospital—Frank Hand, J. W. Crawford.

Mount Zion Hospital—Frank Rodin, Herbert Cohn.

St. Francis Hospital—Constantine Bricca, Aubrey Rawlins.

St. Joseph's Hospital—Roy Parkinson.

St. Luke's Hospital—Ambrose E. Edgerton, Charles Bates.

St. Mary's Hospital—Francis Conlan, Stanley Burns.

Southern Pacific General Hospital—Wilbur Swett.

Stanford University Hospitals—Edward Sewell, Hans Barkan, Harrington B. Graham, Louis Morrison.

United States Marine Hospital—Rea E. Ashley.

University of California Hospitals—Wallace Smith, Fred C. Cordes, Robert C. Martin, C. Allen Dickey.

Veterans Administration Facility—J. J. Caviness, Otto Barkan.

OAKLAND EXECUTIVE COMMITTEE

Oliver D. Hamlin, Chairman

Charles A. Dukes, Vice Chairman

Whitfield Crane, Secretary

Clinical Program Committee—Lemuel P. Adams, Warren B. Allen, Harry H. Appledorn, Frank S. Baxter, Julian J. Benton, Whitfield Crane, Charles A. Dukes, Sumner Everingham, Channing Hall, Oliver D. Hamlin, Henry W. Harding, Harold H. Hitchcock, T. O. Lake, Theodore C. Lawson, Ergo Majors, W. Earl Mitchell, George McClure, Albert Meads, Raymond J. Nutting, Clair Rasor, Dextor N. Richards, Milton H. Shutes, H. R. Smithies, Clifford Sweet, Frank Walsh, Don D. Weaver.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY, GYNECOLOGY, OBSTETRICS, ORTHOPEDICS, UROLOGY, PROCTOLOGY, SURGICAL PATHOLOGY, ETC.

CLINICS IN SAN FRANCISCO HOSPITALS

SAN FRANCISCO HOSPITAL

Monday—Stanford University Surgical Service

Amphitheater A—9

J. W. CLINE. Cholecystectomy. Discussion by H. P. HILL.
C. MATHEWSON. Splenectomy. Discussion by E. WAYBURN.
L. ELOESSER. Lobectomy, demonstration of cases; flap operation for tuberculous empyema, demonstration of cases. Discussion by P. K. BROWN.

Amphitheater B—9

E. MORRISSEY. Section and immediate suture of tibial nerve for ischemic pain, demonstration of cases, lantern slides and specimens.
E. B. TOWNE. Neurolysis.
M. R. OTTINGER. Torek operation for undescended testicle.
C. MATHEWSON. Open reduction for spiral fractures of the tibia.

Amphitheater C—9

R. DUNN. Conization of cervix.
H. VON GELDERN. Perineal repair.
A. V. PETTIT. Vesicovaginal fistula.
K. SCHAUPP. Hysterectomy.
D. DALLAS. Vaginal hysterectomy.

Amphitheater A—2

D. WOOD, H. BARLAND, L. ELOESSER and G. BARNETT. Clinical pathological conference.
E. BUTLER. Treatment of perforated peptic ulcers.
J. W. CLINE. Volvulus of cecum.
M. R. OTTINGER. Injection treatment of rectal prolapse.
W. ROGERS. Volkmann's contracture following burns.

Nurses Home—2

A. V. PETTIT. Treatment of pelvic inflammatory disease.
R. DUNN. Treatment of incomplete abortions.
D. DALLAS. Operations in obstetrics.
G. HARTMAN. Pyuria and hematuria.
L. MICHELSON. Obstruction of the neck of the bladder in the female.

Ward B Solarium—2

J. M. MEHERIN. Fractures of the tarsal scaphoid.
C. MATHEWSON. Open reduction of spiral fractures of the tibia.
D. KING. Functional anatomy and pathology of the shoulder joint.
J. W. CLINE. Internal derangement of radio humeral joint.
N. J. HOWARD. Fracture of the humerus, moving picture demonstration.

Tuberculosis Solarium—2

W. L. ROGERS. Communicating empyemas.
P. PIERSON. Influence of position on respiratory volume.
T. WIPER. Congenital cystic diseases of the lung.
L. ELOESSER. Bronchial obstruction.

Tuesday—University of California Surgical Service

Amphitheater A—9

HAROLD BRUNN. Gastric resection; lobectomy, one stage.
C. M. JOHNSON. Perineal prostatectomy. Discussion by FRANK HINMAN on prostatectomy versus transurethral resection.
C. L. CALLANDER. Amputation of the thigh, a new method.

Amphitheater B—9

GEORGE K. RHODES. Gastro-enterostomy.
WILLIAM G. MOORE. Hysterectomy for leiomyomata of the uterus.
F. S. FOOTE. Ventral hernioplasty, Gallie technique.
ALSON R. KILGORE and OTTO H. PFLUEGER. Radical mastectomy.

Amphitheater C—9

F. C. BOST. Repair of ruptured ligaments of knee joint.
W. J. COX. Reduction and fixation of fracture of hip.
F. C. BOST, W. J. COX and V. M. DILLON. Reduction of several fractures.

Amphitheater A—2

H. E. RUGGLES and J. L. CARR. X-ray and clinical pathological conference.
HAROLD BRUNN. Pulmonary abscess. Discussion of the bacteriology by C. W. WEISS.
C. L. CALLANDER. A new amputation of thigh.
H. M. BLACKFIELD. Skin grafts.

Lecture Room, Nurses Home—2

Symposium: The Newer Bacteriological Aspects of Surgical Infections

C. W. WEISS. Newer investigations on inflammation.
A. HEIM. Staphylococcal and streptococcal infections.
A. P. KRUEGER. The principles of vaccine therapy.
K. F. MEYER. Anaerobic infections—tetanus, gas bacillus, etc.

Social Hall, Nurses Home—2

GEORGE K. RHODES. A study of 1000 cases of acute appendicitis at the San Francisco Hospital.
C. M. JOHNSON. Renal and perirenal infections.
MARGARET SCHULZE. Pelvic inflammatory disease.
K. O. HALDEMAN. Acute osteomyelitis.
S. H. MENTZER. Cholecystitis.
H. J. MCCORKLE. Perforated peptic ulcer.

Assembly Hall, Tuberculosis Wing—2

F. G. LINDE and K. O. HALDEMAN. Compression fractures of the spine.
H. W. FLEMING. Head injuries.
A. L. BROWN. Traumatic injuries of the chest.
GEORGE K. RHODES. Traumatic injuries of abdomen.
F. C. BOST. Fractures of the ankle with ligamentous tears.

Thursday—Stanford University Surgical Service

Amphitheater A—9

D. KING. Arthroplasty of hip joint.
W. ROGERS. Apicolysis, paraffin fill; demonstrations. Discussion by CABOT BROWN.
L. ELOESSER. Thoracoplasty; demonstration of cases. Discussion by P. PIERSON.
T. WIPER. Radical amputation of breast, demonstrations. Discussion by H. GARLAND.

Amphitheater B—9

J. M. MEHERIN. Gastric resection (Billroth I). Discussion by H. P. HILL.
E. BUTLER. Pyloroplasty. Discussion by GEORGE BARNETT and L. ELOESSER.
E. BUTLER. Resection of colon for congenital atresia.
M. R. OTTINGER. Injection treatment of inguinal hernia.

Amphitheater C—9

G. HARTMAN. Suprapubic prostatectomy.
R. G. CRAIG. Nephrectomy.
L. REYNOLDS. Cystectomy and transplantation of ureter.
L. MICHELSON. Plastic of kidney pelvis.

Amphitheater A—2

D. WOOD, H. GARLAND, L. ELOESSER and G. BARNETT. Clinical pathological conference.
J. W. CLINE. Staphylococcus immunity tests.
EMMET RIXFORD. Coccidioidal infections.
N. J. HOWARD. Amebic granuloma bowel.
E. BUTLER. Roentgen aid to diagnosis in injuries to the chest and abdomen.
C. MATHEWSON. Lymphogranuloma inguinale; benign strictures of rectum.

Nurses Home—2

R. G. CRAIG. Ureteral pain of obscure origin.
L. REYNOLDS. Torsion of testicle.
G. HARTMAN. Renal tuberculosis.
K. SCHAUPP. Fibromyoma of uterus.
H. VON GELDERN. Plastic operations of pelvis.

Ward B Solarium—2

C. MATHEWSON. Demonstration of closed methods of treatment of fractures of the extremities.
D. KING. Treatment of sclerosing osteomyelitis.
J. M. MEHERIN. Spontaneous tendon rupture.
E. MORRISSEY. Tumors of the cauda equina as the cause of sciatica.

Tuberculosis Solarium—2

L. SEAPY. Ultimate results of thoracoplasty.
W. L. ROGERS, CABOT BROWN, P. PIERSON and W. BURKHARD. Symposium on surgical treatment of pulmonary tuberculosis.
L. ELOESSER. Treatment of benign esophageal stricture.

Friday—University of California Surgical Service

Amphitheater A—9

H. W. STEPHENS. Thoracoplasty.
S. H. MENTZER. Cholecystectomy.
HAROLD BRUNN. Resection of colon; resection of rectum.

Amphitheater B—9

GEORGE K. RHODES. Choledocholithotomy.
LEON GOLDMAN. Thyroidectomy.
A. L. BROWN. Hernioplasty.
L. P. PLAYER. Nephrectomy.

Amphitheater C—9

A. M. VOLLMER. Uterine suspension.
MARGARET SCHULZE. Vaginal repair.
H. M. BLACKFIELD. Repair of Dupuytren's contracture.
FRANK HINMAN. Uretero-intestinal implantation; radical cystectomy.

Operating Room No. 3—9

H. A. BROWN. Neurolysis.
H. W. FLEMING. Repair of defect of skull.

Ward B—9

F. C. BOST, W. J. COX and V. M. DILLON. Demonstration and reduction of fractures.

Amphitheater A—2

H. E. RUGGLES and J. L. CARR. X-ray and clinical pathological conference.
Fractures of the extremities with associated nerve injuries. F. C. BOST: The orthopedic aspect. H. A. BROWN: The neurosurgical aspect.
C. M. JOHNSON. Traumatic injuries of the genito-urinary tract.
W. J. COX. Rupture of the lateral ligaments of the knee joint.

Lecture Room, Nurses Home—2

GEORGE K. RHODES. Gas bacillus infections.
H. H. ALEXANDER, JR. Treatment of fractures of the os calcis.
HOWARD MORROW and HIRAM E. MILLER. Fungoidal infections.
M. W. DEBENHAM and J. M. SWINDT. Hepatic abscess, amebic and pyogenic.
SIDNEY OLSEN. Tuberculosis of the genito-urinary tract.

Social Hall, Nurses Home—2—Cancer Symposium

OTTO H. PFLUEGER. Presentation of cases from the San Francisco Hospital Cancer Clinic.
ALSON R. KILGORE. Operation for cancer of breast; end-results, demonstration of cases.
LLOYD BRYAN. Radiotherapy of breast cancer.
JESSE CARR and JUSTIN A. WILLIAMS. Bone tumors.

Assembly Hall, Tuberculosis Wing—2

LEON GOLDMAN. Hyperparathyroidism; parathyroid adenoma associated with generalized osteitis fibrosa cystica.
A. M. VOLLMER. Sympathectomy in dysmenorrhea.
WALTER BIRNBAUM. Traumatic injuries of tendons.
C. G. LYON. Fractures of the acetabulum with central dislocation.

SAN FRANCISCO EMERGENCY HOSPITAL
(Auditorium of Public Health Building)

Tuesday—2

JACOB C. GEIGER, Director of Public Health of San Francisco. Hospital care of the indigent.
EDMUND BUTLER. Organization of the emergency hospital service of San Francisco.
GEORGE K. RHODES. Acute operative work in the San Francisco Hospital; handling of the indigent.
JOHN R. SULLIVAN. Treatment of cyanide poisoning.
Inspection of Central Emergency Hospital and the ambulances.

FRANKLIN HOSPITAL

Tuesday—9

G. W. PIERCE and G. O'CONNOR. Plastic surgery.
L. BROOKS. General surgery.
E. GEHRELS. Gastro-intestinal surgery.

Thursday—9

E. GEHRELS. Gastro-intestinal surgery.
L. BROOKS. General surgery.
G. HARTMAN and J. B. LEONARD. Urologic surgery.
J. SALE, V. DILLON and O. MONTGOMERY. Orthopedic operations.

UNIVERSITY OF CALIFORNIA HOSPITAL

Monday—9

R. WARD—Room B. Thyroidectomy.
H. BLACKFIELD—Room B. Plastic surgery.
W. JONES, JR.—Room C. Operation for brachial neuritis.
C. ROSSON—Room C. Ventral hernia.
M. WOOLF—Room D. Rectal surgery.
M. L. MONTGOMERY—Room D. Hernioplasty.
F. LYNCH—Room E. Cervical repair, cystocele, rectocele, suspension, appendectomy.
D. MORTON—Room E. Martin flap cervical resection, cystocele, rectocele.
F. BAKER—10. Demonstration of pool therapy.
JOHN E. MCGUINNESS—11. Demonstration on the use of the Pavaex unit.
ROBERT S. STONE—11:30. Demonstration of deep therapy plant.

Monday, 2—Toland Hall

EUGENE S. KILGORE. Circulatory disease in differential diagnosis of acute abdomen.
H. GLENN BELL and THEODORE ALTHAUSEN. Pre-operative management of cholecystitis, operative mortality without routine glucose, with routine glucose therapy, with Rose-Bengal and when necessary other routine tests as a pre-operative routine.
FRANK LYNCH and ROBERT STONE. Uterine cancer, X-ray therapy, radium therapy, follow-up.
HOMER WOOLSEY. Carcinoma of stomach.
ROBERTSON WARD. Carcinoma of thyroid.

Monday, 2—Cole Hall

HOWARD C. NAFFZIGER and O. W. JONES, JR. Prevention and repair of skull defects.
FREDERIC FOOTE and J. E. CARR. Experimental work on jaundiced patients, clinical application.
HANS LISSER and L. P. PLAYER. Presentation of a boy, aged 9 years, 4 years after the removal of an adrenocortical tumor causing sexual precocity.
H. BRODIE STEPHENS. Subphrenic abscess.
Z. E. BOLIN. Mixed tumors of the parotid gland.

Tuesday—9

F. HINMAN—Room B. Perineal prostatectomy; nephrectomy.
W. I. TERRY—Room C. Substernal goiter.
H. G. BELL—Room C. Esophageal diverticula.
L. C. ABBOTT—Room D. Sacral fusion with bone graft.
F. LYNCH—Room E. Procidentia; vaginal hysterectomy and creation of new pelvic floor.
D. MORTON—Room E. Hysterectomy for fibroids.
N. N. EPSTEIN—10. Demonstration of pyro therapy.
JOHN E. MCGUINNESS—11. Demonstration on the use of the Pavaex unit.
ROBERT S. STONE—11:30. Demonstration of deep therapy plant.

Tuesday, 2—Toland Hall

WALLACE TERRY, H. H. SEARLS and E. I. BARTLETT. Our conception of chronic diffuse thyroiditis.
J. B. DEC. M. SAUNDERS. The knee joint, functional anatomy and surgery.
P. CAMPICHE. The present status of radiotherapy from the surgeon's standpoint.
CARL HOAG. Reconstruction of the bile ducts.
KARL SCHMIDT. The bile salts.

Tuesday, 2—Cole Hall

M. L. MONTGOMERY. Acute intestinal obstruction.
FRANK HINMAN. Uretero-intestinal anastomosis.
F. I. HARRIS and A. C. WHITE. Injection treatment of hernia.
T. F. MULLEN. Antethoracic esophagoplasty.
ROY ABBOTT. The shoulder joint.

Tuesday, 2—Room 437, Clinic Building

Staff. Cancer clinics. FRANK LYNCH: Gynecology. E. I. BARTLETT: Breast. ROBERTSON WARD: Thyroid. H. GLENN BELL: Gastro-intestinal. C. L. CONNOR: Pathology. R. S. STONE and H. RUGGLES: X-ray.

Thursday—9

F. HINMAN—Room B. Cystectomy; uretero-intestinal implantation.
H. C. NAFFZIGER—Room C. Laminectomy for spinal cord tumor.
H. H. SEARLS—Room C. Thyroidectomy.
E. I. BARTLETT—Room D. Radical mastectomy.
W. MOORE—Room E. Hysterectomy for fibroids.
M. SCHULZE—Room E. Cervical repair, cystocele, rectocele, suspension.
F. BAKER—10. Demonstration of pool therapy.
JOHN E. MCGUINNESS—11. Demonstration on the use of the Pavaex unit.
R. S. STONE—11:30. Demonstration of deep therapy plant.

Thursday, 2—Toland Hall

W. J. KERR. The heart in hyperthyroidism.
H. H. SEARLS. Thyroid adenomata with lowered or normal basal metabolic rate.
DANIEL MORTON and ROBERT STONE. X-ray pelvimetry, direct method.
FRANCIS S. SMYTH. Calcium and phosphorus metabolism, clinical applications.
E. I. BARTLETT. Bone tumors.

Thursday, 2—Cole Hall

W. W. WASHBURN. Treatment of acute perforated ulcer.
M. L. MONTGOMERY. Peripheral vascular disease, sheets showing studies and care.
O. W. JONES, JR. and HOWARD C. NAFFZIGER. Low back pain and cord tumors.
FRANK HINMAN and W. K. MURPHY. Prostatism.
DR. REINHARDT. Vitamin C deficiency in arthritis.

Thursday, 2—Room 437, Clinic Building

Staff. Cancer clinics. M. S. WOOLF: Rectum. L. R. TAUSSIG: Skin. R. MILLZNER: Lip and mouth. E. I. BARTLETT: Bone tumors. C. L. CONNOR: Pathology. ROBERT S. STONE and HOWARD RUGGLES: X-ray.

Friday—9

H. G. BELL—Room B. Cholecystectomy.
H. H. SEARLS—Room B. Gastro-enterostomy for pyloric obstruction.
H. C. NAFFZIGER—Room C. Splanchnic resection for essential hypertension.
H. BRUNN—Room C. Lobectomy.
L. C. ABBOTT—Room D. Stabilization of foot; congenital torticollis.
F. LYNCH—Room E. Removal of ovarian cyst and hysterectomy.
D. MORTON—Room E. Cesarean section.
N. N. EPSTEIN—10. Demonstration of pyro therapy.
JOHN E. MCGUINNESS—11. Demonstration on the use of the Pavaex unit.
R. S. STONE—11:30. Demonstration of deep therapy plant.

Friday, 2—Toland Hall

C. D. LEAKE and FRANK LYNCH. Divinyl oxide, a new general anesthetic agent, its clinical use.
M. S. WOOLF. Posterior resection of the rectum and its applied anatomy.
H. GLENN BELL. Benign lesions of the small bowel.
ROBERT AIRD. Encephalography, clinical and experimental.
F. C. BOST. Fracture of the ankle, ligamentous tears.

Friday, 2—Cole Hall

HAROLD BRUNN. Acute empyema.
HIRAM MILLER. Coccidioides.
KARL MEYER. The treatment of tetanus.
ROBERT AIRD and HOWARD C. NAFFZIGER. Intradural injections for intractable pain.
JAMES W. MORGAN. The present conception of fistula and its management.

Daily Demonstrations

C. L. CONNOR. Demonstration in pathological museum.
JOHN B. DEC. M. SAUNDERS and STANFORD V. LARKEY. Exhibit on history of surgery.

STANFORD UNIVERSITY HOSPITAL

Monday—2—Lane Hall

Symposium on Industrial and Orthopedic Surgery

EMMET RIXFORD. Knotty problems in industrial surgery: Traumatic carcinoma of breast; ruptured heart; traumatic thrombosis of iliac and other large veins.
A. L. FISHER. Sacro-iliac lesions.
DON KING. The shoulder joint: Its functional anatomy and pathology.

Symposium on Surgical Maggot Therapy

M. C. MENSOR. Indications, technique and end-results.
EDWIN W. SCHULTZ. Bacteriological studies with relation to bacteriophage.
LEON O. PARKER. Mechanical and chemical aspects of larval therapy.

Monday—2—Nurses' Auditorium

Symposium on Gastro-Intestinal Surgery

A. L. BLOOMFIELD. The physiology of the stomach with reference to surgical indications.
GUNTHER NAGEL. Indications for partial excision of the pyloric sphincter.
EMILE HOLMAN. Important technical considerations in resection of the stomach for carcinoma.
R. A. SCARBOROUGH. Developments in the surgical treatment of carcinoma of the rectum, with a summary of 200 cases treated in the Stanford University clinic during the past twenty years.
ERNST GEHRELS. Problems in surgery of the colon and rectum: Closure of artificial colostomy; preservation of anal sphincter; treatment of diverticulitis.

The Etiology of Chronic Regional Enteritis

GUNTHER NAGEL. Clinical observations.
F. L. REICHERT and M. E. MATHES. Experimental observations.
DAVID A. WOOD. Multiple primary carcinomata complicating multiple polyposis of colon.
NELSON HOWARD. Amebic granuloma of large bowel.

Tuesday—9—Operating Suite

EMMET RIXFORD, P. K. GILMAN, STERLING BUNNELL, EMILE HOLMAN, E. B. TOWNE, F. L. REICHERT, GUNTHER NAGEL, ERNST GEHRELS and R. A. SCARBOROUGH. General, industrial, proctological and neurological surgical operations.

Tuesday—9—X-ray Department

R. R. NEWELL and staff. Demonstration of supervoltage installation; demonstration of irradiation of cancer by divided dosage according to Coutard and Regaud; demonstration of radium application from multiple points to surface of body; follow-up studies on the irradiation treatment of cancer.

Tuesday—2—Lane Hall

LUDWIG EMGE. Dysmenorrhea, causes and treatment; sterility, diagnosis and treatment.
C. F. FLUHMANN, P. E. HOFFMANN and G. F. JONES. Endocrinological aspects of gynecology: Modern methods of diagnosis; blood and urine hormone tests; biopsy of the endometrium; hormone therapy.
A. V. PETTIT. Results of hyperpyrexia in treatment of acute and chronic pelvic inflammatory disease.
LUDWIG EMGE. Radiation therapy of carcinoma of cervix; methods and end-results.

Tuesday—2—Nurses' Auditorium

PHILIP PIERSON. Carcinoma of lung simulating inflammatory disease.
EMILE HOLMAN. Technical improvements in partial selective thoracoplasty; resection of transverse processes; resection of lower third of scapula.
MARY E. MATHES. An experimental study of the effect of various pathological conditions upon the dual blood supply of the lungs; ligation of the pulmonary artery as a therapeutic measure in pulmonary hemorrhage.
EDGAR POTH. A simple apparatus for tidal and siphon irrigation and its application in the treatment of empyema.
DAVID A. WOOD and MARY E. MATHES. Clinical and experimental observations on the dual blood supply of the lungs in various pathological states.

Thursday—9—Operating Suite

LUDWIG EMGE, H. A. STEPHENSON, C. F. FLUHMANN, W. E. STEVENS, GLENN CRAIG, P. E. HOFFMANN, A. D. DAVIS and STERLING BUNNELL. Gynecology, female urology, plastic and industrial surgery, operative clinic.

Thursday—9—X-Ray Department

R. R. NEWELL and staff. Demonstration of consultation stereoscope; demonstration of pelvimetry; demonstration of lipiodol injection of bronchi.

Thursday—2—Lane Hall

A. L. FISHER. Second type arthritis complicating Colles' fracture, fracture of neck of femur, hallux valgus and its treatment.
DON KING. Regeneration of the semilunar cartilages.
LEON PARKER. Injections of joints with local anesthesia for diagnostic purposes with particular reference to the sacro-iliac joint.
NELSON HOWARD. The treatment of epiphyseal fracture, dislocation at elbow.
MERRILL MENSOR. Unusual fractures of the spine.
A. L. FISHER. The treatment of flat feet.

Thursday—2—Nurses' Auditorium

Symposium on Surgical Diseases of the Breast

LUDWIG EMGE. Experimental production of malignancy of breast.
NELSON HOWARD. Etiology of cystic mastitis; experimental reproduction by hormone injection.
ERIC LILJENCRANTZ. Treatment of inflammatory carcinoma of breast.
F. L. REICHERT. Demonstration of cleared specimens of breast tumors—benign and malignant.

Symposium on Surgical Diseases of the Blood Vessels

F. L. REICHERT. Treatment of circulatory disease of the extremities; interruption of the sympathetic nerves; passive vascular exercise.

NELSON HOWARD. The varicose state: Results of operative and injection treatment of varicose veins.

EMILE HOLMAN. The recognition and treatment of an arteriovenous aneurism.

F. L. REICHERT. The "peripheral heart" to produce passive vascular exercise.

Thursday—3:30—Lane Library

F. L. REICHERT. History of Medicine. Demonstration of medical incunabula.

Friday—9—Operating Suite

A. L. FISHER, DON KING, MERRILL MENSOR, LEON PARKER, J. R. DILLON, R. L. RIGDON, LLOYD REYNOLDS, LEO ELOESSER and EMILE HOLMAN. Orthopedic, urologic and thoracic surgery.

Friday—2—Lane Hall

J. R. DILLON. Treatment of chronic pyelitis and pyelonephritis. Technical improvements in surgical treatment of undescended testicle.

W. E. STEVENS. Observations on pathological conditions of the urinary tract in women.

EDGAR POTH. A new aseptic technique for ureteroenterostomy; mechanism of ascending infection of the urinary tract; experimental observations.

W. E. STEVENS and S. P. SMITH. Scientific exhibit of interesting female urological conditions.

Friday—2—Nurses' Auditorium

P. K. GILMAN. Adenomata of the thyroid producing toxic symptoms without elevation of basal metabolic rate.

L. G. DOBSON. End-results of thyroidectomy for Graves' disease over a ten year period; demonstration of case showing cord paralysis coming on three weeks after thyroidectomy.

M. E. MATHES. Results of thyroidectomy for Graves' disease in children.

L. G. DOBSON and L. D. HOWARD. Clinical and laboratory studies in a case of hyperparathyroidism.

F. L. REICHERT. Neuralgias of the cranial nerves, demonstration of patients, lantern slides.

R. A. SCARBOROUGH and E. J. POTH. Important mechanical factors in spinal anesthesia.

DAVID A. WOOD. Exhibit of interesting endocrine tumors; operative and autopsy material.

Friday—3:30—Surgical Laboratory

F. A. FENDER. Demonstration of apparatus for remote excitation of nervous system.

U. S. MARINE HOSPITAL

ROBERT A. JONES. Excision of pilonidal sinus and rectal operations; cholecystectomy and autoplasty (Gillies tubular flap); Dupuytren's contracture.

E. M. TOWNSEND. Inguinal hernioplasty using pedicle fascia strips; operation for sciatica (Heyman); arthrotomy of the knee with excision of internal semilunar cartilage; bunion operation (Peabody); phrenic neurectomy; transurethral resection of prostate.

Staff. Fracture clinic.

U.S.S. RELIEF

Tuesday

HORACE R. BOONE—9. Sacrococcygeal dermoids; caudal anesthesia in rectal surgery; traumatic surgery.

SOUTHERN PACIFIC GENERAL HOSPITAL

Tuesday—9

WALTER B. COFFEY and JOHN D. HUMBER. Superior cervical sympathectomy for angina pectoris (motion picture film in natural colors); surgical treatment of angina pectoris, superior cervical sympathectomy.

C. A. WALKER. Operation for hydrocele with reference to total removal of sac.

C. P. MATHE and T. E. GIBSON. Transurethral prostatectomy.

EARL N. GREENWOOD. Cholecystectomy.

FRANK R. GIRARD. Inguinal hernia, ambulant treatment by injection.

O. F. NOLAN and T. E. GIBSON. Suprapubic prostatectomy.

Tuesday—2

C. A. WALKER. Phrenicectomy for pleurocardiac adhesions; open reduction of fractures.

BERNARD KAUFMAN. Phrenic nerve and precordial pain.

EARL N. GREENWOOD. Fractures of vertebrae, with particular emphasis on treatment, complications, end-results of compressed fractures.

R. EMMET ALLEN. Complicating factors in the management of peptic ulcer.

PHILIP K. BROWN. Peptic ulcer, indications for surgical treatment.

C. A. THOMAS. Surgical measures in pulmonary tuberculosis.

W. W. WASHBURN. Tumors of the parathyroid gland.

HENRY D. BRUSCO. Coccidioides granuloma, diagnosis and treatment.

W. F. SWETT and J. C. WILLIAMS. Clinics and demonstration of cases.

Thursday—9

G. B. O'CONNOR and G. W. PIERCE. Skin graft and pedicle graft in severe burn.

C. A. WALKER. Plating of fracture of tibia.

C. P. MATHE and T. E. GIBSON. Hydronephrosis, plastic repair of, with case reports.

W. W. WASHBURN. General surgical operations: Gastroenterostomy; thyroidectomy.

J. A. GUILFOIL. Injection of hemorrhoids, demonstration.

FRANK R. GIRARD. Ambulant treatment of varicose ulcers, demonstration of cases.

Thursday—2

C. A. WALKER and J. E. BOHM. Mortality rate in operations for appendicitis.

L. B. CROW. Mediastinal effusions, roentgenological studies of; X-ray demonstration of unusual fractures and results of treatment.

W. F. SWETT and J. C. WILLIAMS. Clinics and demonstration of cases.

W. T. CUMMINS. Pathological exhibits, gross and microscopic.

W. W. WASHBURN. Disabilities following fractures; factors influencing period of recovery, demonstration of cases.

J. E. BOHM. Dysinsulinism, hypoglycemia.

HENRY D. BRUSCO. Tularemia.

BERNARD KAUFMAN. Pathological lesions above the diaphragm simulating abdominal pathology.

J. A. GUILFOIL. Chronic appendicitis, results of operations for.

MOUNT ZION HOSPITAL

Monday—9

HAROLD BRUNN—Room 1. Radical mastectomy, cautery.

FRANKLIN I. HARRIS—Room 1. Ileitis, repair of incisional hernia.

H. A. R. KREUTZMANN—Room 2. Nephrotomy, pyelotomy or nephrectomy.

REGINALD K. SMITH, ABRAHAM BERNSTEIN, MORRIS H. SILVERBERG, ZACHARY COBLENTZ and SAMUEL R. SHERMAN—Room 3. Obstetrical clinic, classical cesarean section.

A. LINCOLN BROWN—Room 3. Alcoholization of intestinal nerves for tuberculosis, Leotta method.

ALFRED ZOBEL and DAVID A. SUSNOW—Room 4. Electrocoagulation of tumors of the rectum; hemorrhoidectomy, local infiltration.

EDWARD BOLZE, HERBERT SCHULTZ and SIEGBERT LAZAR—Room 5. Demonstration of induction of anesthesia by avertin, evipal, gas and oxygen, spinal.

Monday—2

HAROLD BRUNN, A. LINCOLN BROWN, JOHN J. SAMPSON and HAROLD ROSENBLUM. Symposium on surgery of the heart with particular reference to adhesive pericarditis.

ALFRED ZOBEL and DAVID A. SUSNOW. Melanosis coli, demonstration of cases.

FRANKLIN I. HARRIS. Treatment of appendix stump with particular attention to the non-inversion method.

FRANKLIN I. HARRIS and ALFRED S. WHITE. Injection treatment of hernia.

LOUIS C. JACOBS, ABELSON EPSTEEN, BERNARD STRAUSS and MAX POLSE. Urological lesions of the female bladder; exhibition of rare pathological entities.

Tuesday—9

FRANKLIN I. HARRIS—Room 1. Torek operation for undescended testicle, first and second stage procedures; subtotal gastrectomy for peptic ulcer; cholecystectomy.

LAWRENCE HOFFMAN—Room 2. Hysterectomy.

FELIX L. PEARL—Room 2. Resection of inferior mesenteric plexus and superior hypogastric plexus for atonic lesion of bowel.

LIONEL D. PRINCE, ABRAHAM SIRBU and DAVID CHARMACK—Room 3. Reduction of fractured os calcis; removal of semilunar cartilage of knee; orthopedic operations; correction of metatarsus varus primus in hallus valgus.

ABELSON EPSTEEN—Room 4. Injection of vas deferens for chronic epididymitis.

BERNARD STRAUSS—Room 4. Hydrocele operation; plastic for phimosis.

MAX POLSE—Room 4. Cystoscopy.

GEORGE W. PIERCE—Room 4. Plastic surgery.

EDWARD BOLZE, HERBERT SCHULTZ and SIEGBERT LAZAR—Room 5. Demonstration of induction of anesthesia by avertin, evipal, gas and oxygen, spinal.

Tuesday—2

FRANKLIN I. HARRIS. Chronic cicatrizing enteritis (regional ileitis), demonstration of cases.

A. LINCOLN BROWN. Collapse therapy in the treatment of pulmonary tuberculosis; pneumococcus peritonitis; rupture of thoracic duct; demonstration of cases.

JOSEPH LEVITIN. Roentgenological demonstration: Localization of lung abscess, carcinoma of lung and tuberculosis.

ALFRED S. WHITE. Treatment of staphylococcus infection with staphloid.

SAMUEL COHN and FRANKLIN I. HARRIS. Treatment of undescended testicle by operation and glandular extracts.

FRANKLIN I. HARRIS. Surgery in acute cholecystitis.

G. Y. RUSK and GERSON R. BISKIND. Demonstration of interesting pathological surgical specimens.

FRANKLIN I. HARRIS and G. Y. RUSK. Sarcoma of breast, pathology.

SAMUEL R. SHERMAN. Traumatic rupture of spleen with recovery; lymphoblastoma (?) of mediastinum in two-year-old child.

Thursday—9

HAROLD BRUNN—Room 1. Total thyroidectomy for cardiac disease; intestinal resection for carcinoma of bowel; thoracic operation (thoracoplasty or lobectomy).

A. LINCOLN BROWN—Room 2. Thoracoplasty; interruption of phrenic nerve; cholecystectomy.

FRANKLIN I. HARRIS—Room 3. Hysterectomy for fibroid of uterus; interposition operation for prolapse; repair of rectal sphincter following complete tear.

HARRY BLACKFIELD—Room 4. Plastic correction of congenitally protruding ears.

HARRY BLACKFIELD and FRANKLIN I. HARRIS—Room 4. Homan's operation for incision and inlay skin graft of chronic leg ulcer.

EDWARD BOLZE, HERBERT SCHULTZ and SIEGBERT LAZAR—Room 5. Demonstration of induction of anesthesia by avertin, evipal, gas and oxygen, spinal.

Thursday—2

HAROLD BRUNN. Pelvic appendicitis followed by carcinoma of rectum.

Staff—Diseases of the peripheral arteries: Determination of vasomotor spasm, peripheral nerve novocaine block, cervicodorsal sympathetic novocaine block; presentation of cases, results of treatment. Conditions amenable to surgery of the sympathetic nervous system: Ganglionectomy for hyperhidrosis, demonstration of case showing results.

EDWARD H. BOLZE. Resumé of 2,000 cases of avertin basal anesthesia; experiences with evipal.

LIONEL D. PRINCE, ABRAHAM SIRBU, DAVID CHARMAK and GEORGE KARFIOL. Fractures of os calcis, 40 cases; replacement of tibial shaft by fibula following osteomyelitis; treatment of bursitis, presentation of cases; clinical demonstrations of orthopedic cases.

CHARLES WEISS, JEAN S. FELDHEYM and DAVID LUBIN. Intraperitoneal vaccination; value of amfetin; experimental pulmonary abscesses in monkeys.

Friday—9

LOUIS C. JACOBS, ABELSON EPSTEEN, MAX POLSE and BERNARD STRAUSS—Room 1. Calculi of the urinary bladder; suprapubic prostatectomy; transurethral prostatectomy.

FELIX L. PEARL—Room 2. Anterior cervicothoracic ganglionectomy; extraperitoneal lumbar ganglionectomy.

FRANKLIN I. HARRIS, MORRIS J. GROPER and ALFRED S. WHITE—Room 3. Blood transfusion, citrate, Linderman and Unger methods.

WILLIAM WALDEYER—Room 4. Appendectomy; hernioplasty.

EDWARD BOLZE, HERBERT SCHULTZ and SIEGBERT LAZAR—Room 5. Demonstration of induction of anesthesia by avertin, evipal, gas and oxygen, spinal.

Friday—2

GEORGE W. PIERCE and HARRY BLACKFIELD. Plastic surgery: Demonstration and exhibition of postoperative plastic results.

ERNST WOLFF. Pyloric stenosis, diagnostic problems; twelve years experience with appendicitis in childhood, no mortality.

ANITA FAVERMAN. Nonsurgical chronic abdominal pain in childhood (adolescence).

ABRAHAM BERNSTEIN. Obstetrical clinic: Pre-eclamptic toxemia and treatment (motion picture); application of forceps and repair of perineum (motion picture); extraperitoneal cesarean section, presentation of cases and lantern slides.

Daily—9 to 12 and 2 to 4

JOSEPH LEVITIN. Exhibit of diagrams and illustrative X-rays—interlobar septa, lobes of the lung, study of the lower lobes, division into superior and inferior parts.

ST. LUKE'S HOSPITAL

Monday

OTTO H. PFLUEGER—9 (Room B). Cancer of lip.

RUDOLPH L. DRESEL, GEORGE J. MCCHESNEY and W. J. COX—9:30 (Room A). Insertion of pin in fracture of the neck of femur.

ALANSON WEEKS and G. D. DELPRAT—10:30 (Room A). Chronic cholecystitis and cholelithiasis.

DUDLEY SMITH and J. M. MORGAN—10:30 (Room C). Carcinoma of the rectum.

Tuesday

G. D. DELPRAT—9 (Room A). Thoracoplasty.

J. M. MORGAN—9:30 (Room B). Pectenotomy; hemorrhoidectomy.

M. VOLLMER—10:30 (Room A). Rubin's insufflation test.

W. J. COX—10:30 (Room B). Internal derangement of knee joint.

Tuesday—2

GEORGE J. MCCHESNEY, W. J. COX and R. L. DRESEL. Fracture of neck of femur; treatment without external splinting.

W. J. COX. Internal derangement of knee joint, rupture of ligaments; treatment of fracture of femoral neck with Smith-Petersen nails.

DUDLEY SMITH and J. W. MORGAN. Carcinoma of rectum.

ALANSON WEEKS and G. D. DELPRAT. Thoracoplasty.

Thursday

PAUL CASTELHUN—9 (Room B). Appendectomy; herniorrhaphy.

W. G. MOORE—9 (Room C). Supravaginal hysterectomy.

ALANSON WEEKS and G. D. DELPRAT—10:30 (Room A). Common duct stone.

Thursday—2

WILLIAM G. MOORE. Endometriosis; fibromyomata of uterus.

A. M. VOLLMER. Rubin's insufflation test; trichomonas vaginalis.

ALANSON WEEKS and G. D. DELPRAT. Common duct stone; hydatid disease of liver; needling subdeltoid bursa; use of glycerin-alcohol in treatment of wounds and infections.

HOWARD W. FLEMING. Subdural hematomata; cerebrospinal rhinorrhea; relief of intractable pain; cranial approach for orbital tumors; craniocerebral injuries.

Friday

H. FLEMING—9 (Room A). Cerebrospinal rhinorrhea.

A. H. ROSBURG—9 (Room B). Plastic on common duct.

ST. FRANCIS HOSPITAL

Tuesday

LLOYD R. REYNOLDS and OSCAR NOLAN—9. Genitourinary operative clinic; prostatectomy.

W. W. WASHBURN—10. General surgery; thyroidectomy.

Tuesday—2

STERLING BUNNELL. Fracture of neck of femur, lantern slide demonstration.

C. A. FOGERTY. X-ray demonstration of lateral view of neck of femur.

CARL HOAG. Reconstruction of common duct.

RAY KISTLER. Review of some of surgical aspects of diaphragmatic hernia, presentation of cases.

THOMAS STODDARD and EDWARD C. BULL. Orthopedic clinic.

A. M. MOODY. Presentation of interesting pathological specimens.

Thursday

GEORGE W. PIERCE and GERALD B. O'CONNOR—9. Plastic operations: Reconstruction of face after burns; rib cartilage transplant to nose; removal of nasal hump; cleft palate; reconstruction surgery of hand.

Thursday—2

EDMUND J. MORRISSEY. Neurological clinic; shift of pineal as an aid in diagnosis of subdural hemorrhage.

W. B. COFFEY and JOHN D. HUMBER. Five year review of research on inoperable and hopeless malignant cases; the chemical and metabolic relation to the endocrine system and differentiation as to the selective causes of types.

GEORGE W. PIERCE and GERALD B. O'CONNOR. Reconstruction surgery after burns, lantern slide demonstration.

OSCAR NOLAN. Renal sympathectomy.

A. M. MOODY. Presentation of interesting pathological specimens.

ST. MARY'S HOSPITAL

Monday—9

T. GIBSON. Nephrectomy.

W. FAULKNER. Bronchoscopic diagnosis of lung abscess.

Tuesday—9

D. SOOY. Surgical intervention for duodenal ulcer.

T. E. BAILLY. Gastric surgery; gastrectomy.

E. BUTLER. Thyroidectomy, discussion during operation. Clinical survey by RALPH REYNOLDS. Pre-operative throat examination by HARRINGTON B. GRAHAM.

E. TOPHAM. Inguinal hernia.

EDMUND MORRISSEY. Sympathectomy for Raynaud's disease.

Thursday—9

R. YOELL. Gall-bladder surgery; cholecystectomy.

C. P. MATHE. Nephropexy for nephroptosis.

G. K. RHODES. Emergency surgery.

P. ARNOT. Repair of extensive perineal tear; obstetrical surgery.

J. LOUTZENHEISER. Orr treatment for delayed bony union.

T. F. MULLEN. Carcinoma of the esophagus.

Friday—9

J. MCCARTHY. Industrial emergency cases.

HOSPITAL FOR CHILDREN

Monday—10

MURIEL E. EDWARDS and CARL L. HOAG (Room I). Thyroidectomy; thyroglossal duct cyst.
GEORGE W. PIERCE and GERALD B. O'CONNOR (Room II). Plastic operations.
LEROY C. ABBOTT and FREDERIC C. BOST (Room III). Spinal fusion for scoliosis.
F. W. CALLISON (Room IV). General surgical operations.

Tuesday—9

H. H. MARKEL (Room I). Orthopedic operations.
ALMA S. PENNINGTON (Room I). Gynecological operations.
H. A. STEPHENSON and D. A. DALLAS (Room II). Gynecological operations.
SIDNEY OLSEN (Room II). Genito-urinary operations.
PHILIP H. ARNOT (Room III). Gynecological operations.
MARY E. MATHES (Room III). Thoracoplasty.
GEORGE W. PIERCE and GERALD B. O'CONNOR (Room IV). Plastic surgery.
EMMA K. WILLITS (Room IV). Gynecological operations.
MURIEL E. EDWARDS and CARL L. HOAG—10:30 (Clinic Room). Hyperthyroidism in children; thyroglossal duct cysts; demonstration of cases.

Thursday—9

GERTRUDE F. JONES (Room I). Gynecological operations.
LOUIS H. BROCK (Room I). Gynecological operations.
GEORGE K. RHODES (Room II). General surgical operations.
A. R. KILGORE (Room II). General surgical operations.
JOHN SPERRY (Room II). Gynecological operations.
LEROY C. ABBOTT and FREDERIC C. BOST (Room III). Tibial leg lengthening.
H. H. MARKEL (Room III). Orthopedic operations.
FRED H. ZUMWALT and S. BARRE PAUL (Room IV). General surgical operations.
ALBERT S. DAVIS (Room IV). Plastic surgery.
MARY E. MATHES—10:30 (Clinic Room). Pulmonary complications following intracranial operations; demonstration of cases and X-rays.

Friday—9

LEROY C. ABBOTT and FREDERIC C. BOST (Room I). Reconstruction for infantile paralysis.
H. A. STEPHENSON and D. A. DALLAS (Room II). Gynecological operations.
PHILIP H. ARNOT (Room II). Gynecological operations.
EMMA K. WILLITS (Room III). General surgical operations.
ALMA S. PENNINGTON (Room III). Gynecological operations.
MARY E. MATHES (Room IV). Operation for undescended testicle.
A. R. KILGORE (Room IV). General surgical operations.

MARY'S HELP HOSPITAL

Monday—9

Staff. Gynecological operations.

Monday—2

H. VON GELDERN. Pelvic reconstruction.
DUDLEY SMITH. Rectal fistula and hemorrhoidectomy, moving picture demonstration.
BEVERLY SIMPSON. Separated placenta.
ADOLF SCHMIDT. Uterine bleeding.
A. S. MUSANTE. Postoperative infections.

Tuesday—9

Staff. Orthopedic operations.

Tuesday—2

M. MENSOR and LEON PARKER. Skeletal traction in treatment of difficult fractures; osteomyelitis, its treatment with maggots.
LEE HAND. Regeneration of peripheral nerves of hand after injury.
C. C. MCRAE. Injuries to small bones of hand.
R. J. MILLZNER, ZERA BOLIN, R. KILE and I. W. THORNE. Squamous and basal cell carcinoma of face and neck, diagnosis and treatment, pathology, radiation therapy, general discussion.

Thursday—9

Staff. Genito-urinary operations.

Thursday—2

ZERA BOLIN. Biopsies and tumor surgery.
JOHN RICKARD. Mixed tumors of the parotid.
EVERETT CARLSON. Carotid body tumors; splenectomy, indications and technique.
W. B. FAULKNER, JR. Treatment of chest injuries.
W. A. CARROLL. Ruptured kidney.
M. VECKI. Renal movability.
R. J. MILLZNER. Parathyroid injury during thyroidectomy.

Friday—9

Staff. General surgical operations.

Friday—2

DUDLEY SMITH. Cancer of the rectum.
FRANK STILES. Present status of varicose vein treatment.

VETERANS' ADMINISTRATION FACILITY

Monday

BERTHEL H. HENNING—9. Hemorrhoidectomy; fistula-in-ano.
PAUL E. JOHNSON—9. Injection treatment of hemorrhoids; injection treatment of varicose veins.
T. P. JONES—2. The disability rating aspects of diseases of the bones and joints in the Veterans' Administration.
S. B. MCFARLAND—2. Unusual X-ray films.

Tuesday

PAUL E. JOHNSON—9. Gastric surgery; biliary surgery.
LEO ELOESSER—9. Thoracic surgery.

Thursday

PAUL E. JOHNSON—9. Nephrectomy; prostatectomy.
BERTHEL H. HENNING and JOHN A. KENNEDY—9. Indirect inguinal hernia, two cases.
A. J. GRECO—2. Outpatient treatment in Veterans' Administration Facilities.
EDWIN E. ZIEGLER. Nutritional origin of cancer.

Friday

PAUL E. JOHNSON—9. Open reduction of fracture of femur; closed reduction of fracture of tibia with Anderson apparatus.
PAUL E. JOHNSON and BERTHEL H. HENNING—2. Demonstration of Bell orthopedic table and Roger Anderson appliances for treatment of fractures.

FRENCH HOSPITAL

Tuesday—9

W. J. HAWKINS, R. F. GRANT and C. L. COOLEY. Gynecological operations.
ASA W. COLLINS. Radical hemorrhoidectomy by author's method.

Tuesday—2

E. J. MORRISSEY. Diagnosis and treatment of subdural hemorrhages.
FRANK A. LOWE. Fractures of humerus, surgical neck, shaft, supracondylar, moving picture demonstration.

Thursday—9

FRANK A. LOWE. Orthopedic operations.
ASA W. COLLINS. Pylorectomy; gastro-enterostomy.

Thursday—2

ASA W. COLLINS. Author's technique of partial resection of stomach, moving picture demonstration.
FRANK A. LOWE. Internal derangement of knee joint.

Friday—2

Symposium: A preliminary report on investigations being carried out on San Quentin inmates to determine what health reactions follow the surgical removal of gingival and paradental infections (pyorrhea).
PAUL J. BOYENS. Outline of plan and its purposes.
ALEXANDER G. BARTLETT. A general health survey of patients with paradental infections.
HILARIO G. MARQUEZ. Studies of the blood picture in paradental infections.
GEORGE F. OVIEDO. Paradental infections as related to prostatic infection.
I. C. GOBAR. Summary of health changes in relation to paradentosis.
LEO STANLEY. Discussion.

ST. JOSEPH'S HOSPITAL

Monday—9

CURTIS E. SMITH. General surgical operations.
A. SONNENBERG and B. J. ROHLFES. General surgical operations.
T. E. GIBSON—Urological operations.
J. W. JONES. General surgical operations.
A. R. KILGORE. General surgical operations.

Tuesday—9

RALPH SOTO-HALL and KEENE O. HALDEMAN. Orthopedic operations.
J. M. MEHERIN. General surgical operations.
HANS VON GELDERN. Gynecological operations.
T. E. GIBSON. Urological operations.

Thursday—9

CURTIS E. SMITH. General surgical operations.
T. E. GIBSON. Urological operations.
FRANK SHEEHY. General surgical operations.
RALPH SOTO-HALL and KEENE O. HALDEMAN. Muscle and tendon injuries about the shoulder, demonstration of cases.

Friday—9

J. M. MEHERIN. General surgical operations.
FRANK SHEEHY. General surgical operations.
EDWARD MORRISSEY. Neurosurgical operations.
WESLEY E. SCOTT. General surgical operations.
A. R. KILGORE. General surgical operations.
HANS VON GELDERN. General surgical operations.

LETTERMAN GENERAL HOSPITAL

Monday—9

MAJ. F. L. COLE. Cauterization of cervix, perineorrhaphy, suspension of uterus, spinal anesthesia, two cases; perineorrhaphy, hysterectomy, spinal anesthesia; inguinal hernia, spinal anesthesia, two cases; appendectomy, spinal anesthesia, two cases.

Tuesday—9

MAJ. B. S. BURNET. Bone graft in humerus, ether anesthesia; open reduction of tibia, spinal anesthesia; excision cartilage of knee, spinal anesthesia.
MAJ. H. S. BLESSE. Electrical resection of prostate, spinal anesthesia, three cases.

Thursday—9

MAJ. F. L. COLE. Hemorrhoidectomy, spinal anesthesia, three cases; colostomy for rectal carcinoma, spinal anesthesia; thoracotomy (empyema), gas oxygen anesthesia.
MAJ. B. S. BURNET. Orthopedic operations.
MAJ. H. S. BLESSE. Electrical resection of prostate, spinal anesthesia, three cases.

Friday—9

MAJ. P. E. DUGGINS. Curettage, linear cauterization of cervix, perineorrhaphy, suspension of uterus, spinal anesthesia; curettage and insertion of Carsten pessary for sterility, gas oxygen anesthesia.
MAJ. F. L. COLE. Cesarean section, gas oxygen anesthesia; cholecystectomy, appendectomy, inguinal hernia, spinal anesthesia.

SHRINERS' HOSPITAL

Monday

S. L. HAAS and staff—9. Open reduction for congenital dislocation of hip joint; extra-articular graft for tuberculous hip; muscle transplantation.

Tuesday

S. L. HAAS and staff—9. Transplantation of trapezius; Sever operation and teres major transplantation; transplantation of biceps tendon.

Friday

S. L. HAAS and staff—9. Lengthening of tibia and fibula; Hibbs fusion of spine for scoliosis; longitudinal osteotomy of tibia.

CLINICS IN ALAMEDA COUNTY HOSPITALS—WEDNESDAY

SAMUEL MERRITT HOSPITAL

WARREN B. ALLEN—9 (Room I). Reconstruction of skull defects, operation and demonstration of cases. Discussion by H. N. ROWELL.

HENNING KOFORD—9 (Room II). Duodenal ulcer.

FRANK R. MAKINSON—9 (Room III). Cholecystectomy. Discussion by A. A. ALEXANDER.

CHARLES A. DUKES and W. F. WILLIAMS—9 (Room IV). Chest surgery; apicolysis--wax plombage; demonstration of thirty cases by this method by pictures and cases. Medical presentation by HAROLD TRIMBLE.

GEORGE G. REINLE—10:30 (Room I). Nephrectomy. Discussion by E. D. GREER.

MARK L. EMERSON—10:30 (Room II). Rectal surgery and presentation of cases.

W. EARL MITCHELL—10:30 (Room III). Pelvic tumor. Discussion by A. M. SMITH.

HAROLD H. HITCHCOCK—10:30 (Room IV). Orthopedic surgery. Discussion by CHARLES B. FOWLER.

W. H. SARGENT—9–12 (Room V). X-ray demonstration and discussion of cases.

ROBERT A. GLENN—9–12 (Room V). Pathological exhibit; demonstration of frozen section technique and specimens.

Dry Clinic, 2–4

H. N. ROWELL, A. M. SMITH, W. H. STRIETMANN, A. A. ALEXANDER, W. S. KUDER, H. GORDON MACLEAN, FLETCHER B. TAYLOR, VERNE G. ALDERSON and HOBART ROBERTS—2. Symposium on pre- and postoperative care: Management of surgical jaundice and stomach cases; diabetes in surgery; traumatic and postoperative pneumonia; cardiac and renal complications; postoperative psychosis; allergy in relation to abdominal surgery; discussion and demonstration of cases.

BERKELEY GENERAL HOSPITAL

Dry Clinics, 9—12

FRANK D. WALSH. Cholecystitis, observations and comments on surgical treatment.

CLAUDE H. CHURCH. Ectopic pregnancy recurring on same side.

WILLIAM W. CROSS. Polycystic kidney; nephrolithiasis; prostatic management.

W. W. REICH. Parathyroid disease, gross specimens and microprojections.

R. G. VAN NUYS. X-ray demonstration and discussion.

F. J. CARLSON. Ollier's chondrodysplasia, case report; clavicle dressing.

CHILDREN'S HOSPITAL

W. W. CROSS—9. Postmortem findings in the kidneys of children, lantern slide demonstration.

CLIFFORD SWEET and EDWIN M. TAYLOR—9. Clinic on undescended testes: Demonstration of postoperative results; discussion of effect of antuitrin S; demonstration of operation.

ALAMEDA SANATORIUM

J. OHANNESON—9 (Room I). Hernioplasty, under local anesthesia; appendectomy.

G. R. BURKE—9 (Room II). Cholecystectomy.

CHANNING HALL—10 (Room I). Resection of stomach.

ALAMEDA COUNTY HOSPITAL

E. N. EWER—9 (Room I). Total hysterectomy; subtotal hysterectomy; discussion of obstetrical service at Alameda County Hospital.

WHITFIELD CRANE—9 (Room II). Carcinoma of stomach. Discussion by FLETCHER B. TAYLOR.

SUMNER EVERINGHAM—9 (Room IV). Extrapleural thoracoplasty; intrapleural pneumolysis; clinic on phrenic interruption and thoracoplasty. Discussion by CHESLEY BUSH.

ALBERT M. MEADS, LLOYD KINDALL, JOHN A. DAUGHERTY, T. I. BUCKLEY and associates—9 (Room V). Perineal prostatectomy, suprapubic prostatectomy, resectoscopic prostatectomy; operations, demonstration of cases, discussion.

W. F. HOLCOMB—9 (Room VI). Arthroplasty of hip.

FRANK H. BOWLES and THEODORE LAWSON—10 (Room II). Carcinoma of rectum.

CLARENCE A. DEPUY—11 (Room I). Gynecological cancer clinic; demonstration of intraspinal alcohol injections and presentation of cases.

H. W. HARDING and DON D. WEAVER—11 (Room II). Carcinoma of colon.

L. B. BARNARD—11 (Room VI). Arthroplasty of shoulder.

LEMUEL P. ADAMS—12 (Room II). Carcinoma of breast.

Dry Clinics, 2–4:30

CHARLES A. DUKES and associates. Cancer clinic.

HAROLD H. HITCHCOCK, N. A. CARY and associates. Traumatic and orthopedic clinic; demonstration of Sweetland cast dryer, Bell table, plaster models, splints, etc.

W. H. SARGENT and C. B. BOWEN. X-ray exhibit and discussion.

GERTRUDE MOORE. Pathological exhibit and conference.

PERALTA HOSPITAL

ERGO A. MAJORS and LYNN FOREE—9 (Room I). Cholecystectomy. Discussion by JOHN B. DE C. M. SAUNDERS and A. P. KRUEGER.

FREDERIC M. LOOMIS, JOHN W. SHERRICK and J. SCOTT QUIGLEY—9 (Room II). Porro cesarean section.

JOHN LOUIS LOHSE and HAROLD P. MALONEY—11 (Room I). Radical mastectomy.

P. N. JACOBSON—11 (Room II). Pelviolithotomy.

HARRY J. TEMPLETON and C. J. LUNSFORD—11 (Room III). Cutaneous malignancies.

Dry Clinic, 9–12

Calcium dysfunction (including parathyroid hyperplasia). JOSEPH D. COATE. X-ray discussion.
PAUL MICHAEL. Pathological consideration.

EAST OAKLAND HOSPITAL

DON D. WEAVER—9 (Room I). Ulcer of stomach.

H. J. TEMPLETON and C. J. LUNSFORD—9 (Room II). Cutaneous malignancies.

O. R. ETTER—9 (Room III). Dry clinic: Surgery in the diabetic patient.

CLAIR RASOR—10:30 (Room I). Prolapse of uterus.

BROOKS STEPHENS—10:30 (Room II). Hallux valgus.

R. S. VAN NUYS—10:30 (Room III). Dry clinic: Visceroptosis and position of viscera in healthy young adults; X-ray demonstration.

ALEXANDER GRIFFITH—11:30 (Room II). Carcinoma of the rectosigmoidal juncture, Lahey technique.

ALTA BATES HOSPITAL

DEXTER RICHARDS—9 (Room I). Cholecystectomy.
DAVID HADDEN—9 (Room II). Perineorrhaphy.
J. DWIGHT WILSON—10:30 (Room I). Ulcer of stomach.
THOMAS O. LAKE—10:30 (Room II). Hysterectomy.
E. SCHULZE HEALD—11:30. X-ray demonstration.
DAVID HADDEN. Demonstration of anatomy of perineum.

COWELL MEMORIAL HOSPITAL

HERBERT EVANS, ROBERT LEGGE, C. A. KOFOID and associates—9. Exhibit and discussion of latest advances in endocrinology; inspection of laboratories and hospital with special reference to the systematic medical care of university students; discussion of laboratory technique with particular reference to amebiasis.

SURGERY OF THE EYE, EAR, NOSE, AND THROAT

CLINICS IN SAN FRANCISCO HOSPITALS

STANFORD UNIVERSITY HOSPITAL

Monday—9

L. T. HUNNICUTT. Pathological studies.
HAROLD FLETCHER. Presentation of cases.

Monday—2

Staff. Operative clinic.

Tuesday—9

DOHRMANN PISCHEL. Demonstration of retinal detachment cases and moving picture of operation.
HANS BARKAN. Demonstration of strabismus cases and moving picture of O'Connor cinch operation; demonstration of cataract cases with discussion of methods of operating.
AVERY HICKS. Demonstration of cases of ocular torticollis.
FRANK RODIN. Cases of congenital and hereditary eye defects.
H. B. GRAHAM. Congenital occlusion of ear with operative correction.
REA E. ASHLEY. Petrositis with spontaneous recovery; retropharyngeal abscess after tonsillectomy, general anesthesia.
HARVARD MCNAUGHT. Complete stenosis of larynx; bilateral intrinsic carcinoma with laryngofissure.

Tuesday—2

HANS BARKAN, DOHRMANN PISCHEL and staff. Cataract, glaucoma and strabismus operations.

Thursday—9

S. V. CHRISTIERSON. Retropharyngeal abscess; osteoma frontal sinus; syphilis of larynx.
C. B. COWAN. Pathological studies.
F. D. FELLOWS. Acute osteomyelitis following intranasal antrotomy.

Thursday—2

Staff. Operative clinic.

Friday—9

J. A. BACHER. Laryngofissure for bilateral paralysis, amyloid tumor of larynx, retrobulbar neuritis in child; relieved by fronto-ethmo-sphenoid operation; ionization in nasal conditions, acute mastoiditis.
A. G. RAWLINS. Epidemic meningitis coincident with acute exacerbation of chronic, purulent otitis.
R. C. MCNAUGHT. Bilateral external fronto-ethmo-sphenoidectomy with pedicle flaps from septum.

Friday—2

HANS BARKAN, DOHRMANN PISCHEL and staff. Cataract, glaucoma and strabismus operations.

UNIVERSITY OF CALIFORNIA HOSPITAL

Monday—9

W. B. SMITH—Room F. Otorhinolaryngology, operative clinic.

Tuesday—9

W. B. SMITH—Room E. Otorhinolaryngology, operative clinic.

Tuesday—2

WARREN D. HORNER, JOSEPH W. CRAWFORD, C. ALLEN DICKEY and DAVID O. HARRINGTON. Ophthalmological operations.
WALLACE B. SMITH, LOUIS MORRISON and EDITH STOKER. Otolaryngological operations.
JOSEPH W. CRAWFORD. Tuberculosis of eye.
C. ALLEN DICKEY. Surgery of the vertical muscles.
DAVID O. HARRINGTON. Contact glasses, practical demonstration.
R. C. MARTIN and STERLING BUNNELL. Injuries and repair of the facial nerve.

Thursday—9

W. B. SMITH—Room F. Otorhinolaryngology, operative clinic.

Thursday—2

WARREN D. HORNER, JOSEPH W. CRAWFORD, C. ALLEN DICKEY and DAVID O. HARRINGTON. Ophthalmological operations.
WALLACE B. SMITH, LOUIS MORRISON and EDITH STOKER. Otolaryngological operations.
JOHN SAUNDERS. Regional anatomy of the mastoid and pathways of infection of the intracranium.
HOWARD C. NAFFZIGER. Brain abscess arising from middle ear and mastoid infections.
WALLACE SMITH. Phlebitis and thrombosis following middle ear and mastoid infections.

Friday—9

W. B. SMITH—Room F. Otorhinolaryngology, operative clinic.

Friday—2

JOSEPH W. CRAWFORD. The eye in diabetes.
C. ALLEN DICKEY. Ptosis, modification of Motais.
DAVID O. HARRINGTON. Tobacco amblyopia and its treatment.

ST. LUKE'S HOSPITAL

Tuesday and Thursday

JOSEPH L. MCCOOL, C. ALLEN DICKEY, A. E. EDGERTON and CHARLES BATES—9. Ophthalmological clinic.

SAN FRANCISCO HOSPITAL

Tuesday—2

WALLACE B. SMITH, L. F. MORRISON, EDITH M. STOKER and A. H. RICE. Radical mastoidectomy, radical antrum, laryngectomy.

W. D. HORNER, J. W. CRAWFOKD and C. A. DICKEY. Ophthalmology.

Dry Clinic

WARREN D. HORNER. External iridectomy; history, uses, technique, advantages.

C. ALLEN DICKEY and JOSEPH W. CRAWFORD. Safety procedures in cataract operations; akinesis, intra-orbital injections, use of lid sutures, pre-operative medication.

WARREN D. HORNER, C. ALLEN DICKEY and JOSEPH W. CRAWFORD. The use of synthetic epinephrin bitartrate in ocular therapeutics.

AUBREY RAWLINS. Osteoma of the antrum; some curious foreign bodies in the lungs; cases of recovery from otitic meningitis, extensive osteomyelitis of the frontal bone.

HARRINGTON B. GRAHAM. Foreign bodies in the lungs; stenosis of the esophagus; cancer of the larynx.

HARRINGTON B. GRAHAM and J. M. WOLFSOHN. Extensive abscess of the meninges.

REA E. ASHLEY. Treatment of tuberculosis of the larynx.

Thursday—9

ROBERT IRVINE, W. E. BORLEY and FRED BOYLE. Shortening of the eyeball for detached retina, cataract extraction and strabismus.

Thursday—2

WARREN D. HORNER, C. ALLEN DICKEY and JOSEPH W. CRAWFORD. Cataract extraction; strabismus operation; ptosis and glaucoma.

SOUTHERN PACIFIC GENERAL HOSPITAL

Tuesday

WILBUR F. SWETT—9. Hess extraction of cataract.

Thursday

WILBUR F. SWETT—9. Retinal detachment.

WILBUR F. SWETT and JOHN C. WILLIAMS—2. Clinics and demonstration of cases.

LETTERMAN GENERAL HOSPITAL

Tuesday

A. E. SCHLANSER and HERBERT PRICE. Tonsillectomies, adenoidectomies, general anesthesia; eye surgery, general anesthesia; strabismus correction, O'Connor cinch shortening and Jamieson recession.

VETERANS' ADMINISTRATION FACILITY

Tuesday

J. J. CAVINISS. Otolaryngological and ophthalmological operations.

Staff. Bronchoscopic examinations.

ST. MARY'S HOSPITAL

Thursday—2

FRANCIS CONLAN, STANLEY BURNS and FRANK HAND. Treatment of posterior sinusitis; blood dyscrasias in relation to the ear, nose and throat; treatment of bilateral abductor paralysis.

FRANK HAND. Radical antrum.

MOUNT ZION HOSPITAL

Tuesday—9

HERBERT J. COHN, EDWARD LIPSETT and JOHN SKAFF. Tonsil operations, local and general anesthesia; dissection, Sluder, snaretome.

Wednesday—2

HERBERT J. COHN, EDWARD LIPSETT and JOHN SKAFF. Symposium on mastoidectomy.

GEORGE S. LACHMAN. Treatment of corneal ulcers.

CHARLES WEISS. Recently developed concepts in immunology and bacteriology of value to the ophthalmologist.

FRANK RODIN. Treatment of acute iridocyclitis.

G. Y. RUSK. Pathological demonstration of various eye conditions.

Thursday—9

Staff. Nasal operations: Submucous resection, electrocoagulation of turbinates, sinus surgery.

Days to be announced

FRANK H. RODIN and GEORGE S. LACHMAN—9. Ophthalmological operations; Cataract, strabismus, plastic on eyelids.

HOSPITAL FOR CHILDREN

Thursday—2

GEORGE N. HOSFORD and AVERY M. HICKS. Extraction of congenital cataract; muscle operations, O'Connor cinch shortening for simple exotropia, simple esotropia, vertical deviations; muscle transplants for external rectus palsy, superior rectus for paralysis of the superior oblique (Jackson's technique), Motais' operation for congenital ptosis; Toti-Mosher operation for occlusion of nasolacrymal duct (with DRS. MARTIN and HOSMER).

Dry Clinic

GEORGE N. HOSFORD and AVERY M. HICKS. Technique for the determination of the hydrogen-ion concentration of tears; the significance of pH of tears in ocular symptoms and treatment; indications for and results of orthoptic training; relation of vertical imbalance of extra-ocular muscles to gastric symptoms, posture, temperament and aptitude for school work and occupation; congenital muscle palsy; motion picture demonstration of O'Connor technique for heterophoria and heterotropia.

MARY'S HELP HOSPITAL

Friday

FRANK HAND—2. Treatment of bilateral abductor paralysis of the larynx.

HAROLD PARKINSON—2. Differential diagnosis of external otitis.

FRENCH HOSPITAL

Tuesday—9

E. C. FABRE-RAJOTTE, VICTOR D'ERCOLE and C. B. COWAN. Ophthalmological and otolaryngological operations.

CLINICS IN OAKLAND HOSPITALS—WEDNESDAY

PROVIDENCE HOSPITAL

A. J. HOWELL—9. Reconstruction of nose.
RODERIC O'CONNOR—9. Muscle shortening; cataract; Curran operation.
GEORGE MCCLURE and NELSON KEELER—9. Esophagoscopy and bronchoscopy.
RANDOLPH SHARPSTEEN—9. Motais' operation.
ROY NELSON—9. Esophagoscopy and bronchoscopy.
BRUCE STEPHENS, JR.—10. Cataract operation.
FRANK BAXTER—10. Ear, nose and throat operations.
MILTON H. SHUTES—10. Ear, nose and throat operations.
ALEXANDER GALBRAITH—10. Ear, nose and throat operations.
W. A. MAGRATH—11. Eye operations.
ALVIN P. WOLD—11. Eye surgery.
F. C. KRACAW—11. Ear, nose and throat surgery.
R. J. NUTTING—12. Eye operations.
P. T. LEFTWICH—12. Ear, nose and throat surgery.
FRANCIS SHOOK—12. Ear, nose and throat surgery.
SYDNEY N. PARKINSON—12. Treatment of acute and subacute paranasal sinus infections, lantern slide and motion picture demonstration.

Dry Clinic, 2–4:30

Staff.—Discussion of operative procedures, demonstration of cases, lantern and motion picture demonstration.